THE NERVOUS SYSTEM

AND

ITS DISEASES.

A PRACTICAL TREATISE ON NEUROLOGY FOR THE USE OF PHYSICIANS AND STUDENTS.

BY

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DISEASES OF THE BRAIN AND CRANIAL NERVES, WITH A GENERAL INTRODUCTION ON THE STUDY AND TREATMENT OF NERVOUS DISEASES.

WITH FOUR HUNDRED AND FIFTY-NINE ILLUSTRATIONS.

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TO

S. WEIR MITCHELL, M.D., LL.D.,

IN RECOGNITION OF HIS GREAT SERVICES TO NEUROLOGY

AND TO MEDICAL AND GENERAL LITERATURE,

AND

AS A MARK OF HIGH PERSONAL ESTEEM,

THIS BOOK IS DEDICATED

BY

THE AUTHOR.

PREFACE.

THE great work of Gowers is the only extensive treatise on nervous diseases in the English language, although excellent manuals of moderate size have been written; and the author has hence been led to believe that a large text-book, including a comparatively full presentation of the many recent additions to the anatomy and pathology of the nervous system, would be in accord with the needs of the profession.

It has been deemed best to make the present volume complete in itself, with bibliographic and general indexes. In addition to an introduction on the study and treatment of nervous diseases in general, diseases of the brain and of the cranial nerves are discussed in full. Should circumstances permit, this volume will be followed by another, which shall include the remaining diseases of the nervous system, insanity, and the medical jurisprudence of both nervous and mental diseases.

The introductory portion of the work comprises a summary of the embryology, anatomy, physiology, and chemistry of the nervous system, and a consideration of the general pathology, etiology, and symptomatology of nervous diseases, with the methods of investigating them and the best measures applicable in their treatment. The introductory chapters have been made full because of what are believed to be the necessities of American students and practitioners.

While the methods of investigation described and the data found in the clinical and pathological portions of the book are based mainly upon experience in private and hospital practice, it has been the aim to make the volume also representative of the best American and foreign work in neurology.

Close attention has been given to the subject of localization with regard to all portions of the encephalon,—the ganglia and other structures at the base of the brain, the cerebellum, the pons, and the oblongata, as well as the cerebrum. Under cerebral localization are given the results of the latest researches and observations, including the discoveries springing from the newer histological methods.

Diseases of the membranes, sinuses, and veins of the brain are considered separately from arterial diseases, which are chiefly focal, as the latter are more conveniently discussed with other focal lesions, such as tumor and abscess. Special attention has been paid to the

subject of encephalitis. As many of the encephalic affections commonly met with are chronic, and the results of residual lesions or secondary degenerations, it has been thought best to consider these by themselves. The usual order in which the cranial nerves are enumerated is not followed in their discussion, affections of the special senses being considered together. The chapter on these affections includes separate sections on the cochlear and vestibular nerves and their diseases. Affections of taste are discussed at length, because they have as a rule been slighted in other neurological treatises.

The nomenclature and terminology advocated by Professor Burt G. Wilder, of Cornell University, have in the main been used, more largely than in any previous practical work on neurology. The reforms advocated by this distinguished anatomist, especially the introduction of mononyms, are deserving of general adoption.

The method of reference in the bibliographic index is novel, and it is hoped will commend itself to the student and the general reader. This index makes it unnecessary to speak here separately of the author's obligations to his *confrères* in this country and abroad. The plan of using captions for each paragraph has been adopted in the hope that it will be of service in the more exact presentation of facts.

The author is greatly indebted to Professor Wilder for direct help and suggestions connected with neural terminology. To Dr. A. C. Peale he has been under continuous obligations during the progress of the work for assistance rendered, including the searching for references and their verification, the reading and criticism of proof, and the modification and supervision of illustrations. Dr. William G. Spiller, by furnishing notes on recent literature, by special criticisms, and by valuable aid in the pathological sections, has placed him under great obligations to his colleague. Acknowledgments are also due to Dr. J. W. McConnell for assistance in proofreading and in the bibliographic work; to Dr. Henry Leffmann for the revision of the section on the chemistry of the nervous system; and to Dr. F. X. Dercum, Dr. James Hendrie Lloyd, and Dr. Charles W. Burr, of the Philadelphia Hospital staff, for opportunities of studying and using cases in addition to those under his own care.

Many of the illustrations are new, and others obtained from different sources have been more or less modified for the particular purposes of the book. Grateful acknowledgments are due to those authors and publishers, both American and foreign, who have accorded the privilege of using their illustrations, either in the original or in modified forms.

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THE
NERVOUS SYSTEM AND ITS DISEASES.

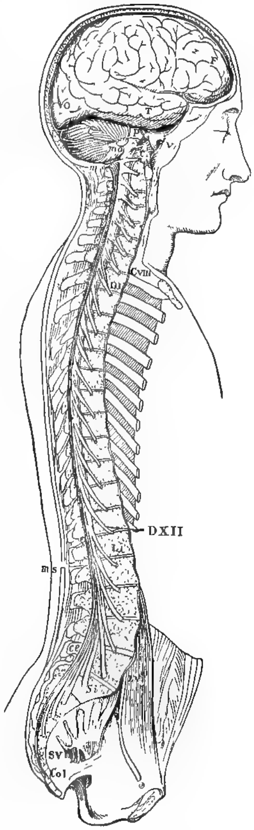
CHAPTER I.

SKETCH OF THE NERVOUS SYSTEM, ITS TISSUES, DEVELOPMENT, ANATOMY, PHYSIOLOGY, NOMENCLATURE, AND CHEMISTRY.

SKETCH OF THE NERVOUS SYSTEM.

The Neuraxis.—Figure 1 shows the general appearance of the neuraxis or encephalospinal axis, half of the skull and trunk being removed. It consists of two united central portions, called respectively the brain or encephalon and the spinal cord or myelon. The cranial and spinal nerves, the roots of which are shown, come to these encephalospinal centres and go from them, carrying, some outward and others inward, the messages which bring man into relation with his environment. The nerves in general constitute the peripheral nervous system, which is represented here only in its beginnings and even in these only partially; it is composed mainly of nerve fibres or conductors which, starting or ending in the neuraxis, ramify to every tissue and organ of the body. Scattered along some of these nerves are small gray masses of nervous matter called ganglia, some at least of which are also centres of energy: so that the central nervous system, while largely within the cranial and spinal cavities, is not strictly confined to them, but exists wherever nervous centres are found; and the peripheral nerves, while chiefly outside, sometimes run a considerable distance within these cavities. The lettering on Fig. 1 indicates some of the most prominent subdivisions of the neuraxis, as follows: F, T, O, frontal, temporal, and occipital lobes of the cerebrum; C, cerebellum; P, pons; *mo*, oblongata; *ms, ms,* point to the upper and lower extremities of the

FIG. 1.

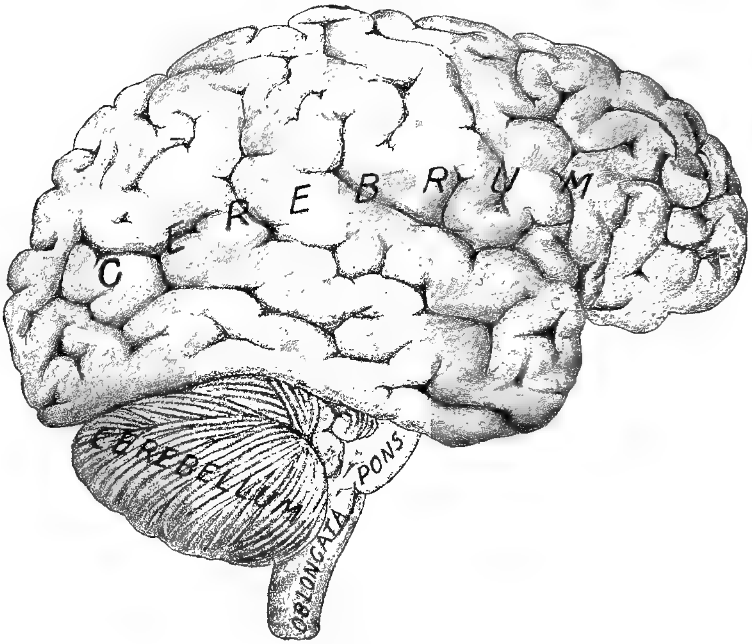


The neuraxis. (Quain's Anatomy, after Bourgery.)

spinal cord ; *ce*, on the last lumbar vertebral spine, marks the cauda equina ; v, the three principal branches of the trigeminal nerve, the largest of the cranial nerves ; CI, the suboccipital or first cervical nerve ; CVIII, the eighth or lowest cervical nerve ; DI, the first thoracic or dorsal nerve ; DXII, the last thoracic or dorsal nerve ; LI, the first lumbar nerve ; LV, the last lumbar nerve ; SI, the first sacral nerve ; SV, the fifth sacral nerve ; CoI, the coccygeal nerve ; s, the left sacral plexus.

Chief Subdivisions of the Human Brain.—The chief subdivisions of the fully developed brain are the cerebrum or great brain, the cerebellum or little brain, the pons, and the oblongata as shown

FIG. 2.

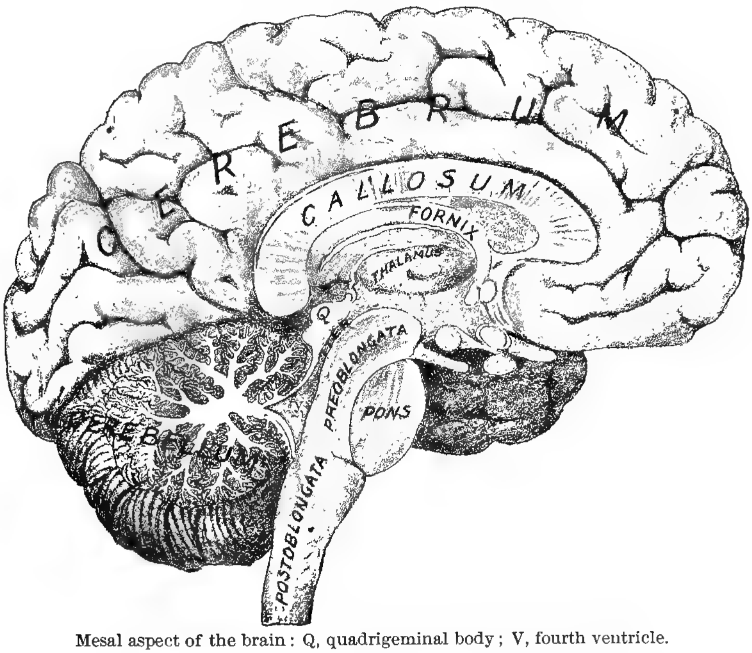


The brain as seen from the right side.

in Fig. 2. The pons and oblongata are more spinal than encephalic in their functions and relations, but from custom and because of their intracranial position are usually regarded as belonging to the brain. In Fig. 3 it will be observed that the oblongata is divided into two portions, the postoblongata and the preoblongata (Wilder), the latter situated mainly between the pons and the cavity of the brain known as the fourth ventricle. The pons, therefore, does not extend through the entire thickness of the neuraxis as represented in most books. Both portions of the oblongata are composed largely of gray deposits or cell nests, while the pons is mainly constituted of nerve fibres or tracts, facts important to remember in connection

with many points to be hereafter considered. The cerebrum and cerebellum are both divided into halves, the hemispheres of the former being separated by a large fissure, and those of the latter by a narrow lobe. If by way of the great fissure which separates the cerebral hemispheres a medisection is made through the callosum, and through the middle lobe of the cerebellum, other parts of the brain can be seen, as in Fig. 3, the most important of them—in addition to the cerebrum, cerebellum, pons, and oblongata—being the callosum, fornix, quadrigeminal body, thalamus, and the iter or aqueduct of Sylvius. The solid bridge of white substance, called the callosum, is the chief connecting link between the two hemispheres.

FIG. 3.



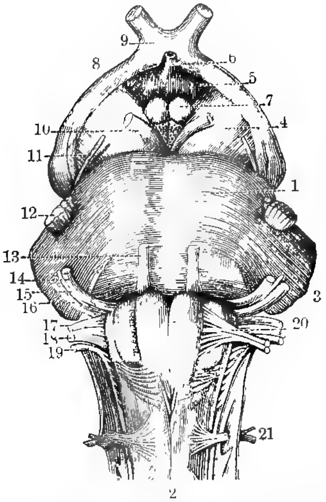
Mesal aspect of the brain: Q, quadrigeminal body; V, fourth ventricle.

The other parts need here only be named. If the brain is cut across it will be found that a gray rind or cortex extends to a slight depth, but that the interior is largely white matter. If sections are made in some positions, as through the prefrontal and occipital regions, or anywhere just below the convolutions, only white matter will be seen. Horizontal sections made more deeply reveal gray masses or deposits, and vertical cuts which divide the cerebral hemispheres, either across or from before backward in their middle portions, show striking gray outlines and contours separated by straits and spaces filled with white matter. These indicate the positions of ganglia and small deposits of gray matter in the brain interior, and also the paths by which some of these bodies are connected with

one another, with the convolutions, and with lower levels of the nervous system.

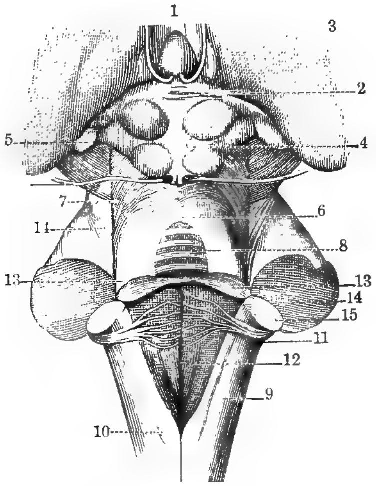
Pons and Oblongata.—The pons and oblongata are intermediate between the spinal cord and both the cerebrum and the cerebellum. Bulb is another name for the oblongata, and hence the phenomena and diseases of this portion of the nervous system are often spoken of as bulbar. Fig. 4 is a representation of the ventral or inferior aspect of these bodies, from which the cranial nerves from the third to the last take their superficial origin. Bifurcating above from the

FIG. 4.



Ventral aspect of the pons and oblongata: 1, pons; 2, oblongata; 3, medipeduncle; 4, crus; 5, tuber cinereum; 6, infundibulum; 7, albicantia, and behind them the posterior perforated space; 8, optic tract; 9, chiasm; 10, oculomotor nerve; 11, trochlear nerve; 12, trifacial nerve, the small or motor root internal; 13, abducent nerve; 14, facial nerve; 15, intermediate portion; 16, auditory nerve; 17, glossopharyngeal nerve; 18, vagus nerve: 19, accessory nerve; 20, hypoglossal nerve; 21, first spinal or suboccipital nerve. (Sappey.)

FIG. 5.

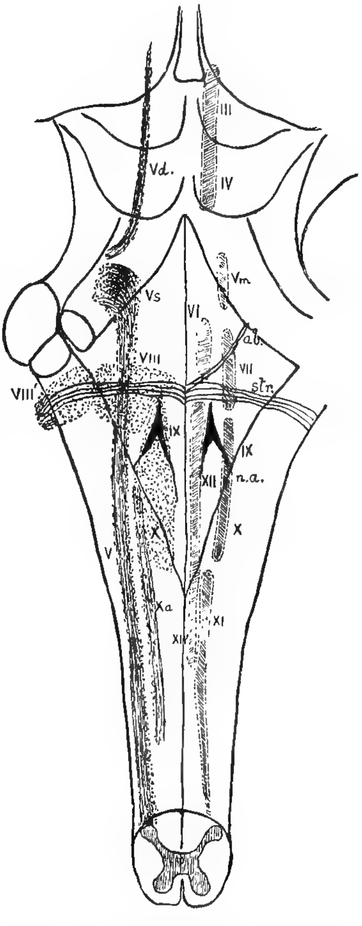


Dorsal aspect of the pons and oblongata, the cerebellum removed: 1, conarium or pineal body turned forward, showing the postcommissure, 2; 3, thalamus; 4, quadrigeminal body; 5, postgeniculum (internal geniculate body); 6, valvula (valve of Vieussens); 7, fillet; 8, lingule; 9, oblongata; 10, postpyramid; 11, restis or restiform body; 12, floor of fourth ventricle; 13, prepeduncle (superior peduncle of the cerebellum) cut through; 14, medipeduncle (middle peduncle of the cerebellum) cut through; 15, postpeduncle (inferior peduncle of the cerebellum) cut through. (Sappey, after Hirschfeld.)

pons are seen the crura, large white cylinders which connect the pons with the cerebrum. Between the crura is a space bounded in front by the chiasm or crossing of the optic nerves; within this space are a gray eminence, the tuber cinereum, prolonged into a hollow funnel-shaped process, the infundibulum, and two white rounded bodies, the albicantia or mammillary eminences; and behind these is a narrow triangular space, the posterior perforated space—to be described later. Looking at the dorsal or superior surface of the oblongata, as in Fig. 5, the fourth ventricle is seen, overhung, when

all parts of the brain are in position, by the cerebellum, as is shown in Fig. 3, page 3. Two pairs of eminences which taken together constitute the quadrigeminal body are also seen; on each side of them the postgeniculum or internal geniculate body; and in front the conarium or pineal body, with portions of the thalami, connected by a band of fibres called the postcommissure. Other parts in the illustration are the valve of Vieussens, a lamina of white matter forming a roof to the upper portion of the fourth ventricle; the fillet, a white band constituting one of the important tracts between the spinal cord and the brain; the lingule, a small process of gray matter in the cortex of the cerebellum; the prepeduncle, the medipeduncle, and the postpeduncle, stout bundles of fibres which connect the cerebellum with the brain above and with the oblongata and the cord below; and the posterior pyramids and restiform bodies, columns uniting the spinal cord and oblongata with the parts above. If transections were made through the pons and oblongata, an intricate arrangement of gray and white matter would be revealed, indicating important nerve centres and fibres. If a horizontal section is made lengthwise, close to the floor of the fourth ventricle, the situation of the nuclei or sources of most of the great cranial nerves will be shown, as in Fig. 6.

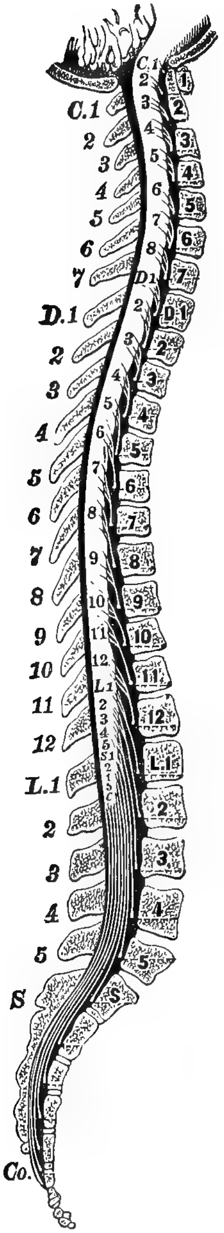
Fig. 6.



Chief nerve nuclei in oblongata, motor nuclei on the right side only, sensory on the left: twice the natural size, seen from behind. The numbers indicate corresponding nerves. V and Vd, spinal and mesencephalic roots of the fifth nerve; Vs, its sensory, and Vm, its motor nucleus; VIII′ is the outer or ventral nucleus of VIII; n. a. is the nucleus ambiguus, an accessory nucleus to IX and X. These nuclei will be considered under cranial nerves. Str, the acoustic striæ. (Quain's Anatomy.)

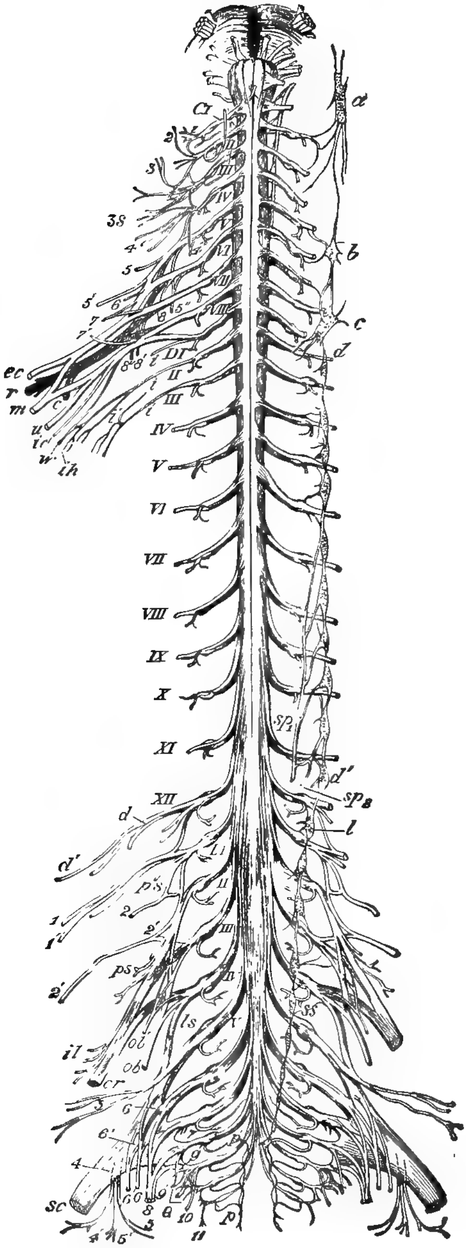
The Spinal Cord and its Nerves.—The adult spinal cord reaches from the oblongata downward to a thread of nerve substance called the filum terminale; from the upper boundary of the first cervical to the second lumbar vertebra, as shown in Fig. 7. It averages about seventeen to eighteen inches in length, while the spinal canal to the last lumbar vertebra is about

FIG. 7.



Relations of the spinal nerves to the bodies and spinous processes of the vertebræ. The positions of the letters and numbers clearly indicate the parts. (After Gowers.)

FIG. 8.



Encephalospinal and gangliated nerves. (Ferrier, after Quain.)

twenty-three inches, and if the sacral portion and the coccyx are included, four inches more should be added. In general it is cylin-

drical in shape, but in two places, at the cervical and lumbar enlargements, where arise the great nerves which go to the limbs, its size is much increased. Below it has a tapering extremity, which is called the conus. Nerves are given off at all levels, and pass through foramens to go to various parts of the body, nearly all descending more or less to these foramens, so that the lower the segment of the cord the longer is the intramural course of the nerves. The lowest spinal nerves form a bundle or leash, named the cauda equina, around and below the conus. Thirty-one pairs of spinal nerves are given off in all—eight cervical, twelve thoracic or dorsal, five lumbar, and six sacral. On transection, the spinal cord, like the brain, reveals an arrangement of gray and white matter, the relative amounts of gray and white substance varying according to the position at which the cut is made, and corresponding differences in the general bulk and form of the cord are also seen. Fibres isolated or in groups in various places cross the white matter to reach the gray. In this gray matter are situated the centres for important functions.

Gangliated System of Nerves, or So-Called Sympathetic Nervous System.—Erroneous views long prevailed as to the gangliated nerves, or so-called sympathetic nervous system, which is simply an arrangement of true spinal nerves connected with a series of ganglia through which they sometimes pass, and which are subdivided according to location. On each side of the ventral aspect of the spinal column is a chain of them united by a longitudinal cord, constituting the lateral ganglia or the vertebral portion of the sympathetic; they are in number, three cervical, twelve thoracic, and four lumbar. Within the cranium are numerous special ganglia all having branches of communication with the cranial nerves. The cervical spinal ganglia also have cranial prolongations. On or near the great viscera of the chest, abdomen, and pelvis is another series of collateral ganglia or plexuses, sometimes called the prevertebral portion of the sympathetic. Besides these are numerous special plexuses, as the cardiac, pharyngeal, laryngeal, pulmonary, coronary, suprarenal, renal, mesenteric, ovarian, and vesical; and the ganglia on the posterior spinal roots really belong to this system of nerves, although they are not usually so regarded. Even if, as held by Paterson, the gangliated nerves are developed from a continuous rod of mesoblast, lying on either side of the aorta, and are only secondarily united with the cerebrospinal nerves, they become parts of one great whole, not an independent system.

Relations of the Spinal Cord to the Limb, Trunk, and Gangliated Nerves.—The relations of the spinal cord to the nerves which go to the trunk and limbs, and to the gangliated nerves, are well represented in Fig. 8. On the left side of the diagram is shown the manner in which the nerves come together to form the brachial or arm plexus or network, and the lumbar and sacral plexuses for

the lower limbs. On the right of the figure is seen the gangliated cord, a series of ganglia united together, and also, an important fact, joined to the spinal cord. The letters indicate certain special ganglia. The spinal nerves are indicated by the Roman numerals. Those below not specially numbered are the sacral nerves. The brachial plexus is composed of branches from Cv to DI, with some communicating branches from CIV and DII; the lumbosacral plexus derives its branches from LI to the fourth sacral nerve inclusive. The individual branches of these plexuses are indicated by letters and small numerals, but need not here be named in detail. The gangliated cord is seen on the right side, with its junction with the spinal nerves: *a*, the superior cervical ganglion; *b*, the middle cervical ganglion; *cd*, the inferior cervical ganglion, united with *d*, the first thoracic or dorsal ganglion; sp_1, the great splanchnic nerve; sp_2, the lesser splanchnic nerve; *d′*, the eleventh thoracic or dorsal ganglion; *ss*, the upper sacral ganglion.

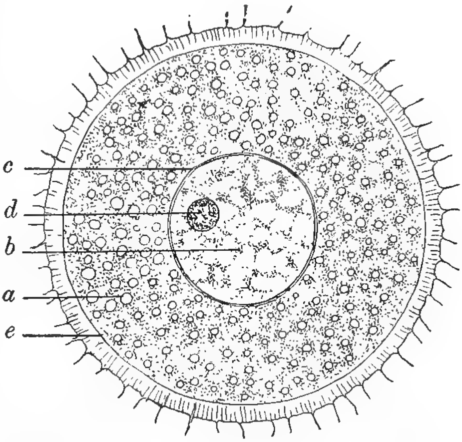
Somatic and Splanchnic Nerves.—Gaskell has proposed the terms somatic and splanchnic in describing the two great varieties of nerves to which reference has just been made. The somatic are the sensory and motor nerves from the dorsal and ventral roots. They supply the chief body or skeletal structures, such as the skin, subcutaneous tissues, muscles, and bones. The term splanchnic is commonly used to describe the great splanchnics, the lesser, and the smallest or renal splanchnic of the ordinary anatomical textbooks; but Gaskell uses it in a more general sense. The term is derived from a Greek word meaning "entrail," and everything pertaining to the viscera is in a generic sense splanchnic. The splanchnic nerves go to the bloodvessels and viscera. Gaskell divides the efferent splanchnics into nerves of the vascular muscles, both vasomotor and inhibitory; nerves of the visceral muscles, both visceromotor and inhibitory; and glandular nerves. Vasomotor nerves for all parts of the body arise in the central nervous system, and are controlled from a centre in the oblongata. In the ventral roots of all the spinal nerves from the second thoracic to the second lumbar, they can be traced as fine medullated fibres, and outward to the visceral ganglia, where they lose their medulla, and are distributed to the heart and vessels, some being reflected to the membranes and vessels of the cord itself. The spinal accessory, or eleventh cranial nerve, acts partly as a somatic and partly as a splanchnic nerve for parts not supplied by the thoracic nerves. The afferent or sensory splanchnic nerves follow closely the efferent distributions, entering the neuraxis in three groups—the highest in the head and neck, the middle between the first thoracic and first lumbar segments, and the lowest from the fifth lumbar to the fourth sacral segment.

NERVOUS TISSUES.

Elementary Tissues.—The elementary tissues of the human body are the epithelial, the connective, the muscular, and the nervous. Our concern is chiefly with the last. The ultimate elements of nervous tissues are called cells, but between the nerve cells and their processes, and separating them from other tissues, are intercellular substances.

The Cell.—An animal cell is nearly always of microscopic dimensions. Every cell consists of protoplasm and a kernel or nucleus; and the nucleus often contains one or several smaller bodies called nucleoli. Sometimes the cell is bounded by a limiting membrane called the cell wall; the nucleus is surrounded by a nuclear membrane. Even the protoplasm of the cell has been shown to be far from simple in composition, containing at least two especially important substances, the spongioplasm and the hyaloplasm. A typical cell is shown in Fig. 9.

Fig. 9.



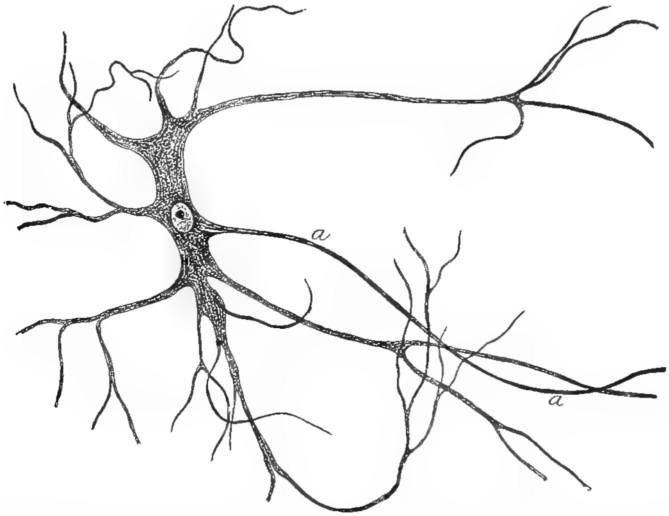
Typical cell,—ovum of cat: *a*, protoplasm; *b*, nucleus; *c*, nuclear membrane; *d*, nucleolus; *e*, true cell wall. (Piersol.)

Nerve Cells.—Nerve cells are found everywhere in the masses of gray nervous matter, small or large. It is important to have a clear idea of their shape, size, composition, varieties, and interrelations; of the tissues which surround them and divide them from one another; of their method of development; of the manner in which they receive, store, reshape, and transmit impressions; of the plan on which they are grouped for special purposes in particular localities; of the extrinsic and intrinsic lesions to which they are liable; and of the possibility of their regeneration after complete or partial destruction. Every nerve cell is to be regarded anatomically and physiologically as a separate unit, in the sense that each with its prolongations has a distinct individuality; that each has a certain work to do in originating, transferring, or using force, although many may act together towards some general end, or one cell or set of cells may even to a certain extent compensate for the destruction of others. It has usually been taught that nerve cells and nerve fibres are different structurally. Strictly speaking, this is not true; they are parts of the same histological unit, as has been especially shown

by recent investigations; but it is necessary for practical purposes to consider separately many of the facts relating to them.

Shapes and Sizes of Nerve Cells.—The shapes and appearances of nerve cells under the microscope differ according to the magnifying power used, the special variety of the cell, the age of the individual, and the disease if a lesion is present. Methods of preparing and staining also cause differences in appearance. The cells of the cerebral cortex, and of other parts, vary according to the region and function subserved; some are ovoid, others of spheroidal or of a nearly round shape; some are angular; others are irregular; some are with and others without visible processes or prolongations, some are spindle-shaped and some flask-shaped. Cells

FIG. 10.



Nerve cell from the spinal cord, isolated by maceration and teasing; the numerous branched protoplasmic processes are somewhat displaced and distorted, owing to manipulation: *a*, axis cylinder process. (Piersol.)

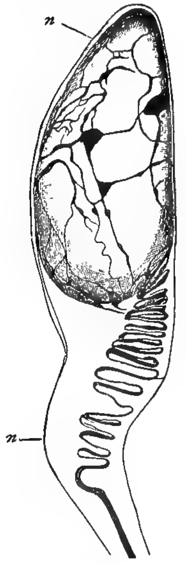
vary widely in size. The nerve cells of some lower animals can be seen even without the microscope. In the lobus nervi vagi of the torpedo, in the oblongata of the river lamprey, and in the spinal cord of the electric eel, are enormous ganglion cells which can be seen with the naked eye (Edinger). As studied in the human body, nerve cells usually range in size between one three hundredth and one three thousandth of an inch. Doubtless large and small cells have differences in function which are to some extent indicated by their size. Of the cells in the motor regions Hughlings Jackson has suggested that small muscles are represented by small cells, or more properly, in his own language, movements which require little energy for the displacements they have to effect are represented by small cells. In the motor subcortex the axis cylinders differ greatly in

size. In the regions of the brain and spinal cord now universally regarded as motor, ganglion cells of large size and pyramidal shape abound.

Structure of the Nerve Cell.—Each nerve cell (Fig. 10) has its distinct kernel or nucleus and within this a smaller kernel or nucleolus. Every nerve cell has one or more processes or prolongations, although these may be broken off or not apparent in some preparations. Views regarding nerve cells have undergone great modifications within a few years, owing chiefly to the investigations of Golgi, His, Ramón y Cajal, Retzius, Koelliker, Nansen, Biederman, Waldeyer, Lenhossék, Marchi, Obersteiner, Van Gehuchten, Sala, Schäfer, Piersol, and Baker. The processes of nerve cells are of at least two kinds. The chief prolongation, and the one first to appear in the development of the nerve cell, is called the axis cylinder, and if but one process is present it is always this. It is continuous with the nerve fibre; the process becomes the fibre or the fibre the process, according to the point of view. The nerve cell has, although not invariably, other short free projections, which have most frequently been called protoplasmic processes. Golgi regarded them as rootlets by means of which the cells were fed from the neighboring gray matter. It is now believed that they are not solely if at all concerned with nutrition, but transmit impulses of some description. Axis cylinders have usually been regarded as composed of fine threads embedded in a clear substance having the appearance of protoplasm, but new views are appearing with reference to these ultimate fibrils. According to Schäfer, it is not improbable that they are excessively fine-walled tubules filled with fluid. In a section of nerve fibre studied by him, the fibrils appeared distinct from one another and tubular in structure, and each showed a minute circular area bounded by a definite line, which he interpreted to represent a minute tubule; and he holds that this theory accounts for the varicosities which appear in broken axis cylinders treated with osmic acid and other reagents. This question is important with regard to the manner in which nerve impulses are conducted.

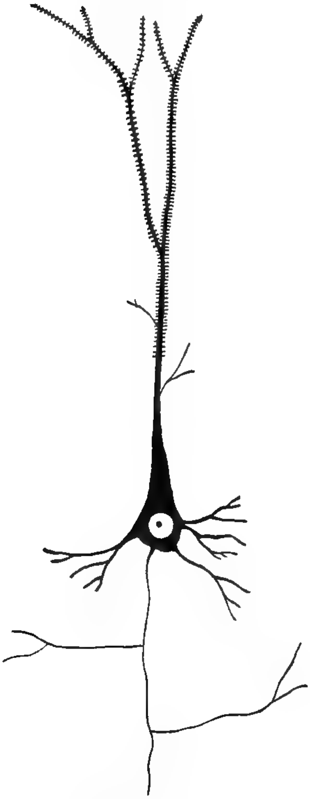
Types of Nerve Cells.—A plain distinction between nerve cells is into two types, one possessing long axis cylinder processes, and the other short axis cylinders. The long process cells may send their processes from the central organ to the periphery as motor, sensory, or secretory fibres, or as long commissural or association fibres. The cells with short or local processes reach only as far as gray deposits or masses in their immediate neighborhood. Golgi believed that the cells with long processes were motor, and that the smaller cells, with relatively short processes, were sensory or receptive, but this distinction has not been confirmed. The process sometimes assumes an exceptional form, as where it becomes spiral in shape as shown in Fig. 11.

FIG. 11.



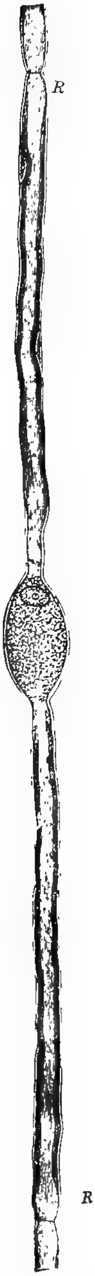
Nerve cell from a "sympathetic" ganglion of the frog, showing the tortuous course and terminal network of the spiral fibre: *n*, neurilemma continued as a delicate sheath. (Retzius.)

FIG. 12.



A projection cell of the cerebral cortex (Schäfer).

FIG. 13.

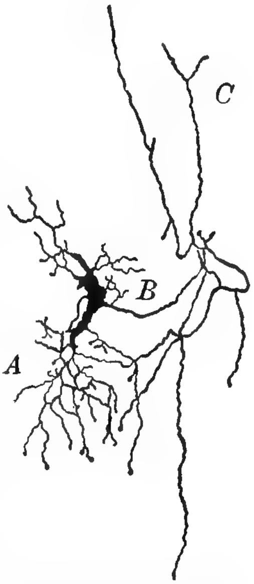


A bipolar nerve cell invested by the primitive sheath: *R*, *R*, nodes of Ranvier. (Quain's Anatomy, from Key and Retzius.)

Cellular Terminology.—The axis cylinders have been designated by various names, as *neuraxons*, *axons*, and *neurons* (Schäfer); the short free projections, or so-called protoplasmic processes, are called *dendrons* (Schäfer), from the Greek δένδρον, a tree. The latter were designated by His as dendrites. Sometimes an axis cylinder process or neuraxon unites with a dendron, and for such combination the name *neurodendron* is used. All nerve cell processes have, at a longer or shorter distance from their origin, a tree-like or tuft-like termination, although this arborization is sometimes difficult to determine. In the cortex dendrons or protoplasmic processes pass upward from the apex of the pyramidal cells through the superficial layers and form a plume-like expansion or *panicle* (Fig. 15), so called because it has the appearance of a pyramidal inflorescence. Nerve cells

are usually named according to the number of their processes, as unipolar, bipolar, multipolar, etc.; but it has been suggested by Schäfer to distinguish them as *dendric* and *adendric*, according as they have or have not dendrons, and according to the number of their neuraxons, as *mononeuric*, *dineuric*, *trineuric*, *polyneuric*, etc. Some of these varieties of cells are seen in Figs. 10, 11, 12, 13, and 14. The term *neuron* has unfortunately been used with several meanings. Wilder, in 1884, proposed it as a designation for the cerebrospinal axis, but has since abandoned it in favor of neuraxis; Waldeyer, in 1891, suggested that it be used to express the idea of a complete nervous unit consisting of the nerve cell, the nerve process, its collaterals, and its termination or end branchings. This use of the word is attractive and has some advantages in teaching, but it seems to me best, following Schäfer, to include under the general term nerve cell not only the body of the cell, but also its processes, collaterals, and endings. As employed by Waldeyer, neurons are spoken of as of the first order and of the second order, but for these designations the terms *projection cell* and *intermediary cell* (Fig. 14) can be substituted. The axis cylinders show a branching network of fibres; their first branches at right angles, which have been particularly demonstrated by Ramón y Cajal and Koelliker as everywhere present in the nervous system, are called *collaterals*. The nerve process or fibre ends in a tuft or brush-like arrangement called the *end brush*. The term *nerve tree* has been aptly applied to the entire branching system of fibres, well shown in Figs. 12, 15, 16, 17, and 18. *Cellulipetal* and *cellulifugal* have been suggested by Ramón y Cajal as expressive of the method of transmission of nerve impulses.*

FIG. 14.



An intermediary nerve cell from the posterior horn of the spinal cord: *A*, dendrons; *B*, neuraxon, soon ramifying, and its branches ending either simply or in bifurcations, as at *C*. (Ramón y Cajal.)

Functions of the Nerve Cell.—The most important function of

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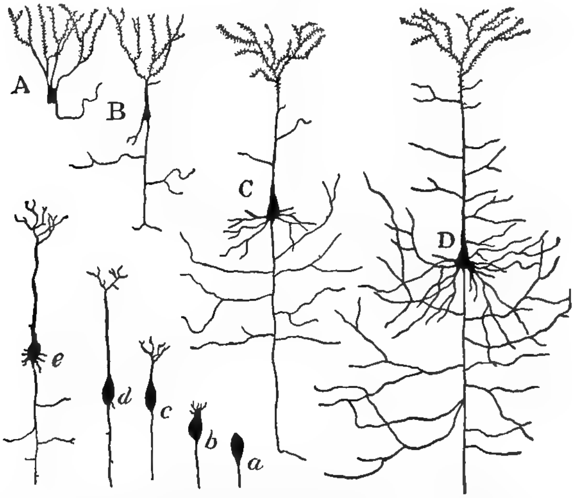
* The suggestion of Waldeyer has found so much favor that it should be understood by the student that the word "neuron" is now most commonly used to mean the cell unit. *Neuraxon*, first proposed by Koelliker, is now largely used for axis cylinder process. *Neurite* is another term for it, suggested by Fish and Rauber. Cellular terminology is still undergoing modifications. Baker has recently suggested *neure* for the nerve cell, including all its appendages; *rhizoneures*, for the cells which form nerve roots, etc. Instead of neuron or neure, Fish has proposed for the cell with its appendages the term *neurocyte*, and *spongiocyte* for the glia or neuroglia cells.

the body or *corpus* of the cell is to preside over the nutrition of the cell and all its processes. If a cell process be separated from the corpus it will eventually die. A nerve cell may originate impulses, but this is probably always by stimulation either from within or from without. The cell and its processes are undoubtedly conductors of impulses; but the connection of nerve cells with each other is physiological and not anatomical; it is by means of processes with processes or of processes with cells. The gray color of the granular looking substance which forms a large part of the central nervous system, and which has been called by Leydig the *punktsubstanz*, or dotted substance, is due to the interblending and interlacing of innumerable nerve cells with their ramifying processes. The isolated

FIG. 15.

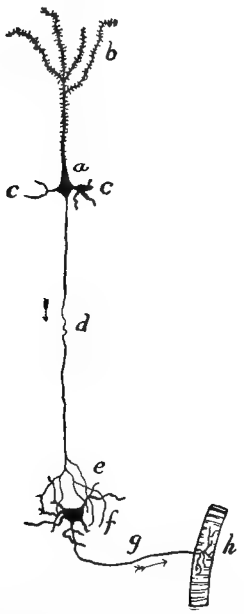


The upper series of cells shows the "psychic" cell in different vertebrates: A, frog; B, newt; C, mouse; D, man. The lower series shows the stages of growth of a single cell: *a*, neuroblast with axis cylinder process just commencing; *b*, panicle commencing; *c*, panicle and axis cylinder process more advanced; *d*, collaterals of axis cylinder appearing; *e*, collaterals of the cell body appearing. (Ramón y Cajal.)

anatomical elements form chains which may be either simple, as in some of the lower forms of animal life, or extremely complicated, as in the central nervous system of man (Schäfer). In Fig. 15 the lower series shows the stages of growth from a cell with a short single process to the full nerve tree. All nervous conduction is from nerve cell to nerve cell by contact. The crossing and recrossing of sensory and motor impulses in the cord and elsewhere are by means of commissural cells, and not by continuous fibres, these cells having short cylindrical processes by which connections are made with other cells. Even the white columns of the cord, although composed largely of vertical fibres, give off at short intervals collaterals with their tree-like formations, and these communicate by

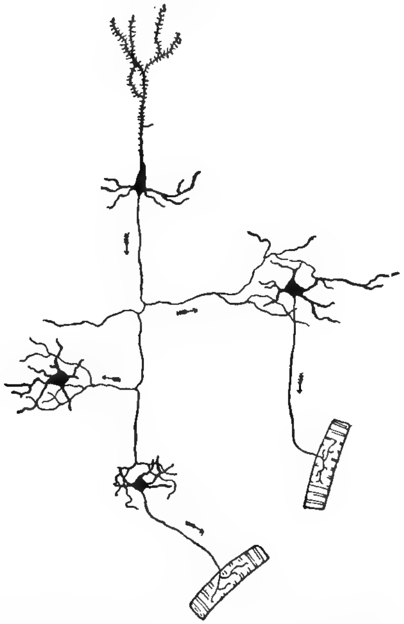
contact with other cells and fibres. Although always fundamentally the same, the process of transmission by nerve cells is often over a long distance and may be elaborate and complicated. A series of intermediary cells may be interposed between the nerve cell which receives the impression and the final destination of the impulse. In Figs. 16, 17, and 18 are representations of the method in which the cells of the cerebral cortex, to which Ramón y Cajal has given the name of *psychic* cells, are united with lower levels of the nervous system and with each other. Fig. 16 shows the chain from the cell and its panicle on the surface of the brain to the musculature. The axis cylinder of the cortical cell extends through the pyramidal tract to a cell in the anterior horn in the spinal cord, embracing the latter with its terminal tufts, the spinal cell with its process and end brush

Fig. 16.



The simplest form of connection of cortical cells: *a*, cell with its processes, *b*, *c*, *d*; *b*, cortical panicle, cellulipetal; *c*, *c*, lateral processes; *d*, axis cylinder process forming a portion of the pyramidal tract; *e*, terminal filaments embracing *f*, a cell of the spinal cord which forms an intermediary cell, sending an axis cylinder process, *g*, in the anterior root of the spinal cord to break into terminal filaments within the motorial plates of a muscle, *h*. (Ramón y Cajal.)

Fig. 17.



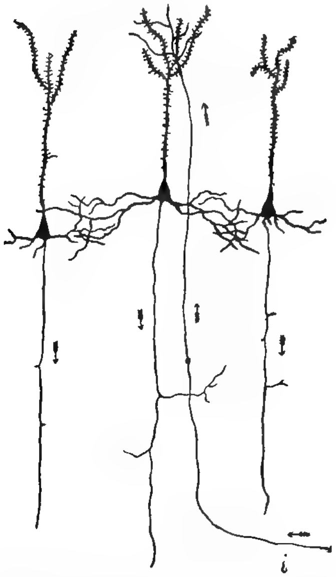
Connection of a cortical cell with more than one intermediary cell (secondary neuron of Waldeyer) by means of the collaterals of its axis cylinder process. (Ramón y Cajal.)

then reaching out to the muscle. In Fig. 17 another method of connection is shown; a lateral and its collaterals from the neuron or axis cylinder process are brought in contact with the branches of another nerve tree. Fig. 18 shows several "psychic" cells connected with each other by the contact of their collaterals. At *i* is represented

the mode by which stimuli are carried by cellulifugal process to the panicle of the cell.

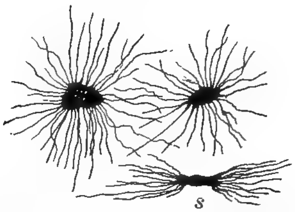
Neuroglia.—Nerve cells are embedded in a substance, usually called neuroglia, found everywhere in the central nervous system, existing in abundance in the brain and spinal cord, among the nerve elements. It is composed of cells and their processes, and is not a true connective tissue, as is indicated by its development from the epiblast and not the mesoblast, although to a certain extent it serves to bind together and shield the more important nervous matter. While essentially a cellular tissue, its cells (Fig. 19) differ from true nerve cells.

Fig. 18.



Connection of cells with each other by means of their collateral processes. Communication of stimuli by means of cellulifugal process *i*, coming up from the zona radiata. (Ramón y Cajal.)

Fig. 19.



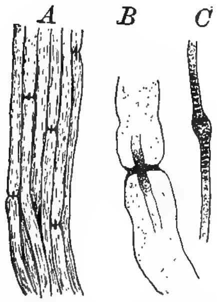
Neuroglia cells, one of which is seen in profile (*s*). Golgi staining.

They have received various names, as glia cells, spider cells, or Deiters's cells. The neurogliar network is really composed of branchings of these cells loosely or compactly arranged. In the spinal cord they accommodate themselves closely to the nerve fibres, and have been compared to packing material between parallel glass tubes. In other places they have a dense brush-like arrangement. The epithelial cells which line the cavities of the nervous system send out prolongations which blend with the neurogliar tissue, and processes of the pia pass into the brain and cord in various places, these together constituting the supporting or sustentacular tissue of the nervous system. Many of the so-called degenerations of the nervous system, as the scleroses, are usually regarded as primarily affections of the neuroglia, a subject which will be hereafter considered. Bevan Lewis and others have attached great importance to the lymph connective system and the neuroglia in the pathogenesis of mental and nervous diseases; calling the cells of the neuroglia scavenger cells, because they assist in carrying off effete, irritative, and destructive material, disease interfering with their cleansing and

purifying work. They may be overdeveloped or underdeveloped; they may be inflamed or destroyed; or they may insidiously degenerate.

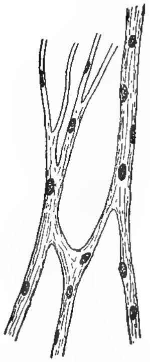
Nerve Fibres.—Nerve fibres, as has been shown, have their origin in nerve cells, usually directly from a ganglion cell the axis cylinder of which passes straightway into the fibre. They vary widely in size, the figures as usually given being from one twelve thousandth to one fifteen hundredth of an inch, and at least three distinct sets are recognized by their differences in size. Most of the somatic cerebrospinal nerves are of large diameter; the gangliated spinal nerves are comparatively much smaller; while fibres of intermediate size are present in large numbers in several of the cranial nerves and in the anterior roots of the spinal nerves. Two principal varieties of nerve fibres are described, the medullated, double bordered or white fibred (Figs. 13 and 20); and the pale gray, non-

FIG. 20.



Silvered nerve fibres: *A*, small bundle of medullated fibres displaying the silver crosses at several nodes; *B*, node of Ranvier under high power; *C*, silvered axis cylinder, showing a biconical enlargement and transverse markings or lines. (Piersol.)

FIG. 21.



Nonmedullated nerve fibres from the gangliated system, the nucleated fibres joining to form a plexus. (Piersol.)

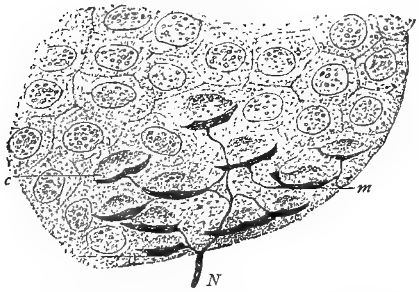
medullated fibres, sometimes called the fibres of Remak (Fig. 21). The medullated fibres are found everywhere in the brain and spinal cord; the nonmedullated chiefly in the gangliated nerves, but both are to some extent present in all parts of the nervous system.

Characteristics of Medullated and Nonmedullated Nerve Fibres.—The medullated nerve fibres differ somewhat in different situations, but usually consist of three distinct parts or structures, a central axis cylinder or axial fibre, a soft inner medullary sheath called myelin, and a firmer outer sheath, the primitive sheath or sheath of Schwann, called the neurolemma or neurilemma. In the brain and spinal cord this latter sheath is usually absent, the medullated fibre here consisting simply of axis cylinder and myelin. The sheath of Schwann is present in the peripheral nerves. The axis

cylinder is enclosed in a tough envelope, which Kühne and Ewald call the keratoid sheath. In peripheral nerves this sheath not only surrounds the axis cylinder but is reflected on the inner aspect of the sheath of Schwann, thus serving also to enclose the myelin. In the peripheral medullated nerves Ranvier discovered here and there dents or breaks in the continuity of the white substance or myelin, which are called nodes, and the subdivisions of the nerve fibre formed by them internodes, or interannular segments (Fig. 20). These appearances are made very plain by certain methods of preparing and staining. Osmic acid makes clear the segmentation, and the constricting bands are readily stained by nitrate of silver. An important point to remember is that the subdivision of a nerve fibre always takes place at a node. The inner nodes vary in length, in the large nerves averaging about one millimetre. Nonmedullated fibres, or the fibres of Remak, are pale, transparent, and faintly striated. They have nuclei believed to be part of a delicate homogeneous sheath which is difficult to make out and is similar to the sheath of the medullated fibres. Unlike medullated fibres, the nonmedullated divide and anastomose, forming a network, although their continuations are similar.

Nerve Endings.—Sensory nerve endings are grouped into tactile cells, tactile corpuscles, and end bulbs (Piersol). Fig. 22 is a representation of a special method of nerve ending within the epidermis. A compound tactile cell or corpuscle results from the union of two simple tactile cells. The end bulbs are a special group of nerve terminations generally cylindrical in type, and usually composed of three parts, a capsule, an inner bulb, and the nerve fibre. They are of various forms, which of course cannot be fully considered in a neurological textbook. The nerves of voluntary muscles usually terminate in a special form of nerve expansion to which the name motorial end plate has been applied. These nerves form plexuses in the muscles, and their fibres divide and subdivide very minutely, passing finally to the sarcolemma. Figures 23 and 24 represent the muscular ending of the nerve fibre of a lizard. Tendons have a nerve supply, although this is not as complete as that to the skin and to the muscles. These nerves to tendons will be of interest in the subsequent discussion of the tendon phenomena, such as knee jerk and ankle clonus. Thus it has been found that a

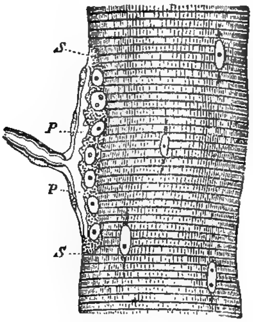
FIG. 22.



Special nerve endings within the epidermis; gold preparation: *N*, nerve fibre entering the epithelium and dividing into the fibrils which are connected with the tactile disks (*m*); upon these latter rest the tactile cells, *c*. (Ranvier.)

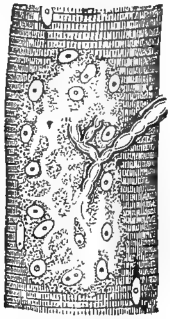
nerve is distributed to the patella and that this has its origin in a certain segment of the lumbar spinal cord. In Fig. 25 is shown the organ of Golgi in the tendo Achillis. Where the tendons unite with the muscles are spindle-shaped bundles into which medullated fibres pass, their axis cylinders dividing and spreading out between the smaller tendon bundles and forming a branched expansion. Various obscure peripheral nervous affections will probably ultimately find their explanation through a more intimate knowledge of the nerve terminal structures, their functions, and the methods by which they can be morbidly influenced. Wedenski makes a suggestion which may prove fruitful, namely, that the rhythmical variations in muscular contractions may be due to the storing of energy in nerve endings. Nerves end differently in different organs, as in the skin,

FIG. 23.



Nerve ending in muscular fibre of a lizard. (Highly magnified.) End organ seen edgeways: *s*, sarcolemma; *p*, *p*, expansion of axis cylinder. Beneath this is granular protoplasm containing a number of large clear nuclei and constituting the "bed" or "sole" of the end organ. (Quain's Anatomy, from Kühne.)

FIG. 24.



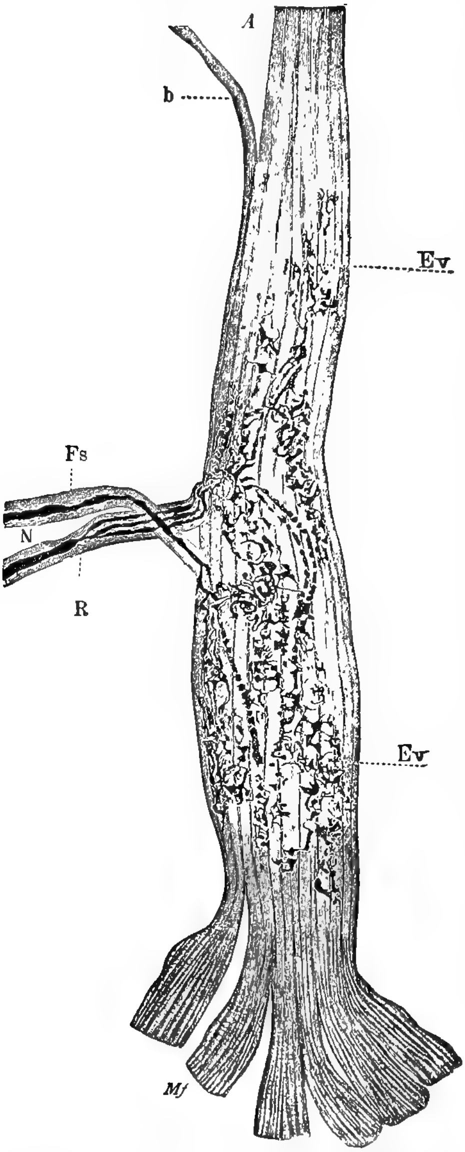
Nerve ending in muscular fibre of a lizard. (Highly magnified.) End organ seen from the surface. The expansion of the axis cylinder appears as a clear network, branching from the divisions of the medullated fibre. (Quain's Anatomy, from Kühne.)

muscles, connective tissues, tendons, bones, vessels, tongue, stomach, uterus, and genital apparatus, according as they are sensory, motor, vasomotor, secretory, inhibitory, etc.; and they connect or anastomose with each other in varying degree according to their peculiar functions and the parts to which they are distributed.

Motor and Sensory Cells.—In the spinal cord in the anterior horns are found cells of very large and of smaller size, ending in a single process which gives off lateral branches; also cells of the anterior and lateral columns, some with few but others with many branches; and cells in the posterior horn, whose axis cylinders do not extend beyond the gray matter but form in it delicate ramifications. In the cerebrum and in the cerebellum at least six varieties of nerve cells have been described. Under appropriate headings, the various properties and peculiarities and the greater abundance of

certain types of cells in different portions of the nervous system will be considered. Nerve cells have been differentiated into at least two great classes according as their work is sensory or receptive or is motor or emissive; and subdivisions of sensory and motor cells have been made according to the manner in which these functions are specialized. According to Golgi, Marchi, and other investigators, the multipolar cells which have been considered can be divided into two types, one motor, in which the neuraxon or axis cylinder becomes a fibre directly; the other sensory, in which the process has a shorter course and passes into a network or complex ramification of processes out of which the nerve fibre seems to arise. It is universally admitted that cells of the first class are motor in function, although they may also have other functions, but the question of the sensory type may be regarded as unsettled. Ramón y Cajal, for instance, believes that the so-called sensory cells of Golgi, which are found in all the layers of the cortex, but chiefly in the fourth, are associative rather than sensory in function. True sensory nerve cells are found in the spinal ganglia, their processes reaching to the integument. It is worthy of consideration whether the cortical termination of the

Fig. 25.

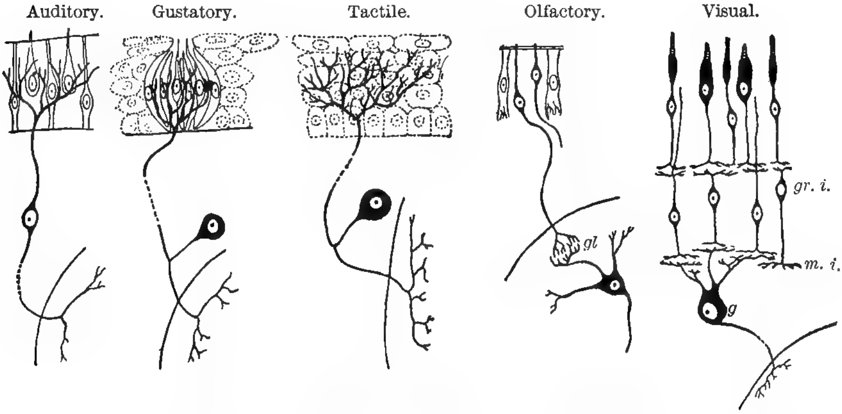


Golgi's corpuscle or tendon spindle from the human tendo Achillis; gold preparation: *N*, nerve fibres surrounded by the perineurial sheath (*Fs*) spreading out into the reticular ramifications (*Ev*) of the axis cylinder; *A*, the tendon bundles, one of which is separated at *b*; *Mf*, the muscle fibres; *R*, node of Ranvier. (After Ciaccio.)

sensory apparatus is not by processes rather than by cells; but it seems necessary to have separate sensory areas with special cells in the higher levels of the brain.

Nervous Structures of the Sense Organs.—The universally admitted senses are those of touch, hearing, sight, smell, and taste. Lenhossék and Retzius have shown that in the earthworm all sensory nerve cells are situated in the epidermis, and that these cells have neuraxons and dendrons which ramify after the manner of such processes in the nervous system of higher animals. It has been demonstrated that the earthworm responds to touch, sound, and light, and probably to odors, although it has no differentiated special sense organs. The different sense organs of the higher animals are therefore probably all modifications of one fundamental ancestral structure. Sensations of touch, hearing, and smell are received by peripheral processes. The peripheral process of the tactile cell passes to a special end organ between the elements of the skin and other structures, and thus the centripetal transmission begins and proceeds. The nerve endings for the auditory and gustatory organs originate from bipolar or unipolar cells placed somewhere along the course of the nerves as shown in the figures. These peripheral nerve cells

FIG. 26.



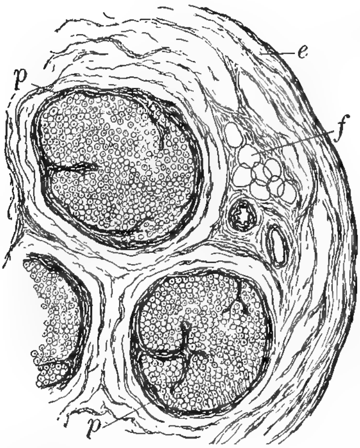
Nerve cells of special sense: *gl*, olfactory glomerules; *g*, ganglion cell of retina; *gr. i.*, inner granules of retina; *m. i.*, inner molecular layer of retina. (Schäfer, modified from Retzius.)

are essentially the same as all other nerve cells. Visual and olfactory impressions are received either directly by the body of the cell or by its very short process, and from the other end fibres pass off to terminate in a tree-like arrangement whose branches interlace with other arborizations, and thus from cell to cell the impulse is conveyed brainward. From morphological standpoints the retina and olfactory bulb are really parts of the brain, in some lower animals the latter forming a large projection from the hemisphere. In the embryo, the retina has the appearance of a stalklike outgrowth

turned inward from the older part of the forebrain. The nervous apparatus of the retina is of a highly specialized character. Some of its great cells correspond to the projection cells of the cortex, with long axis cylinder processes. Retzius has shown with regard to the terminations of the auditory nerve in the semicircular canals and in the organs of Corti, that the true nerve terminals are between the epithelial elements as in other regions. In Fig. 26 are diagrammatic representations of the nerves of special senses, showing their origin, course, and peculiar methods of termination. These in some details bear a striking resemblance to the cells and processes both of the cerebrum and of the cerebellum.

Nerve Trunks or Peripheral Nerves.—What is usually spoken of as a nerve trunk, a nerve, or a peripheral nerve, consists of a collection of cylindrical fasciculi or bundles of white fibres. The entire nerve, as the facial or sciatic, is enclosed in a sheath of connective tissue, which is known as the *epineurium*. Each of the constituent bundles of the nerve is enveloped by its own membranous sheath, the *perineurium*, a more compact mass of connective tissue than the epineurium. Even the nerve fibres which form the separate strands or fasciculi of the nerve are each surrounded by a fine envelope of connective tissue, the *endoneurium* (Fig. 27). These enclosing and separating constituents of the peripheral nerve, although not essential parts of the nerve fibres, must be constantly borne in mind in considering diseases of the nerves; as in some instances these affections are jointly of the connective sheaths and partitions, and of the nerve tissue, or they may be of either one or the other. Nerves are either afferent, incoming, or centripetal; or efferent, outgoing, or centrifugal. The incoming or afferent nerves convey to the centres impressions of various sorts, as of pain, touch, heat, cold, location, pressure, weight, space, sight, hearing, smell, or taste. Some of them simply awaken the latent forces of the spinal cord or other nerve centres, and are hence sometimes called excitoreflex nerves. The efferent or outgoing nerves may be purely motor or muscular; or they may be vasomotor, going to vessels; secretory, supplying glands; or trophic, regulating nutrition.

Fig. 27.

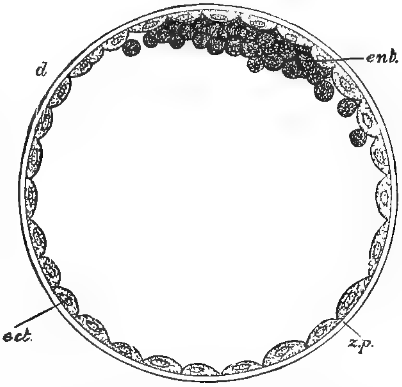


Section of portion of a nerve trunk, including three bundles, or funiculi, surrounded by the perineurium (*p*); the funiculi, together with the bloodvessels and adipose tissue, are united by the more general epineurium (*e*); the sections of the individual nerve fibres are held in place by the endoneurium; *f*, fat cells, near which are the sections of bloodvessels. (Piersol.)

DEVELOPMENT OF THE NERVOUS SYSTEM.

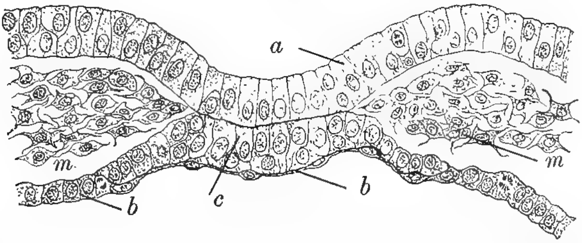
The Blastodermic Layers and the Neural Canal.—After the earliest changes in the fertilized ovum, the blastoderm (Fig. 28), which accumulates over the inner surface of the vesicle, is at first composed of two layers, but eventually of three, which are known as (1) the epiblast, ectoderm, or outer layer; (2) the mesoblast, mesoderm, or middle layer; and (3) the hypoblast, entoderm, or inner layer (Fig. 29). The entire nervous system—brain, spinal cord, peripheral nerves, and ganglia—is derived from the epiblastic layer, which is epithelial, and from which are derived also numerous epithelial structures, and the muscular fibres of the sweat glands. The components of the nervous system are in fact modified epithelial cells. We need only concern ourselves further with that portion of this subject which relates to the neural canal. After the ovum has assumed its hollow spherical shape, a groove or furrow forms on its surface near the primitive groove. This is the first faint streak of the nervous system, the neural or medullary groove, which slowly deepens, and then coming

Fig. 28.



Blastodermic vesicle, its wall formed of a layer of flattened cells, *ect*, with a patch of dark granular cells, *ent*, adhering to it at one part; *z.p.*, zona pellucida. (Quain's Anatomy, after E. v. Beneden.)

Fig. 29.

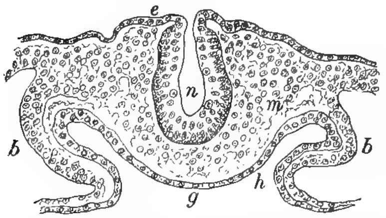


Blastodermic layers of rabbit embryo: *a*, ectoderm; *b*, entoderm; *c*, entodermal cells; *m*, mesoderm. (Piersol.)

together at its edges forms the medullary canal. Fig. 30 and Fig. 31, from the embryo of a rabbit, show the groove just before and just after it has closed. The neural canal is really formed by an infolding of the exterior surface. It begins to close in the posterior cephalic region. It is lined at first with a single layer of columnar epithelial cells; but after a time large round cells, called the germ cells of His, appear.

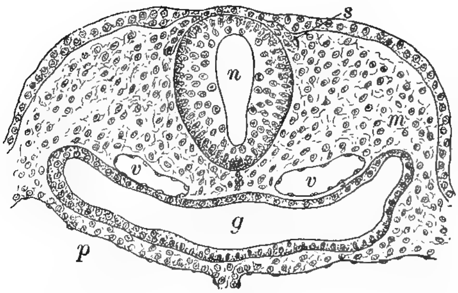
Neuroblasts and Spongioblasts.—It has been shown that the two chief tissues of the nerve centres are the nerve cells and the neuroglia; also that the two main sets of nerve fibres are those of the ventral, anterior, or motor roots and those of the dorsal, posterior, or sensory roots. The development of these tissues and of these roots will now be briefly considered. The germ cells of His actively develop, soon becoming pointed and then pyriform (Fig. 32); and after a time an extended process starts out, giving each cell a tadpole appearance (Fig. 33). In these young cells, called *neuroblasts,* various changes take place, and soon they become towards their centres rod-like, and in contact at their extremities, at the periphery forming a dense network. The cells thus shaped and arranged are known as *spongioblasts* (Fig. 34), the entire reticulated or lattice-like tissue now present being sponge-like in appearance and hence called the *myelospongium;* and in this myelospongium three distinct zones may

Fig. 30.



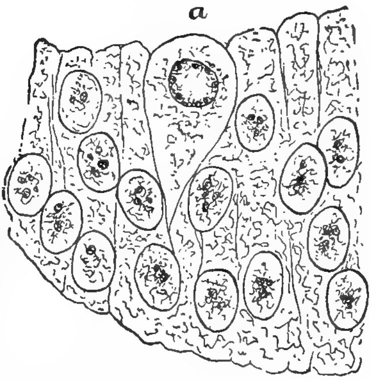
Section of nine-day rabbit embryo, showing open neural tube: *e*, ectoderm invaginated and thickened within neural canal (*n*); *m*, mesoderm; *b*, body cavity; *g*, still open gut, lined by entoderm (*h*). (Piersol.)

Fig. 31.



Section of ten-day rabbit embryo, showing closed neural tube: *n*, neural canal; *s*, area from which segmental ganglia develop; *m*, mesodermic tissue; *g*, gut tube; *v*, *v*, primitive aortæ; *p*, pleuro pericardial cavity. (Piersol.)

Fig. 32.

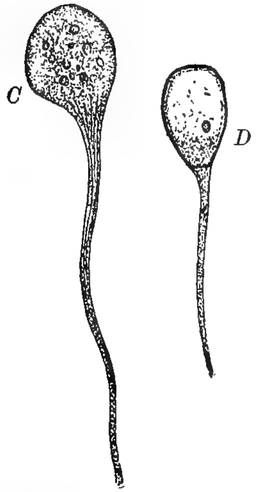


Development of neuroblasts. Epithelium from the medullary canal of the rabbit soon after it has closed. Between the epithelial cells lies a germ cell (*a*) which has become pyriform. (His.)

be made out, an inner towards the central canal, an outside network, and an intermediate zone of nuclei. From the *internal* or *limiting membrane* afterwards develops the substantia gelatinosa centralis, the zone of clear ground substance surrounding the central canal; from the intermediate zone, known as the *columnar* or *mantle layer*, the

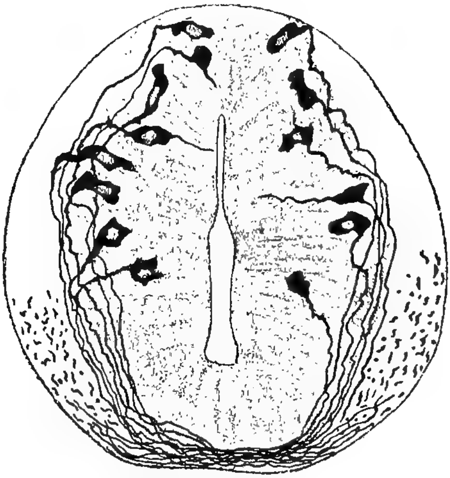
neuroglia of the gray matter; and from the outside reticulum or *velum confine,* the neuroglia of the white matter.

FIG. 33.



C, neuroblast from the embryo of a salamander; *D*, neuroblast from an embryo trout. (His.)

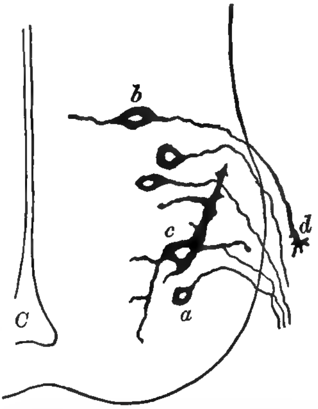
FIG. 34.



Neuroblasts from the spinal cord of an eleven-day chick. Some of the cells show the first formation of the dendritic processes directed towards the central canal. Lenhossék, who at one time regarded this as primary, now looks upon it as a secondary formation. (After Lenhossék.)

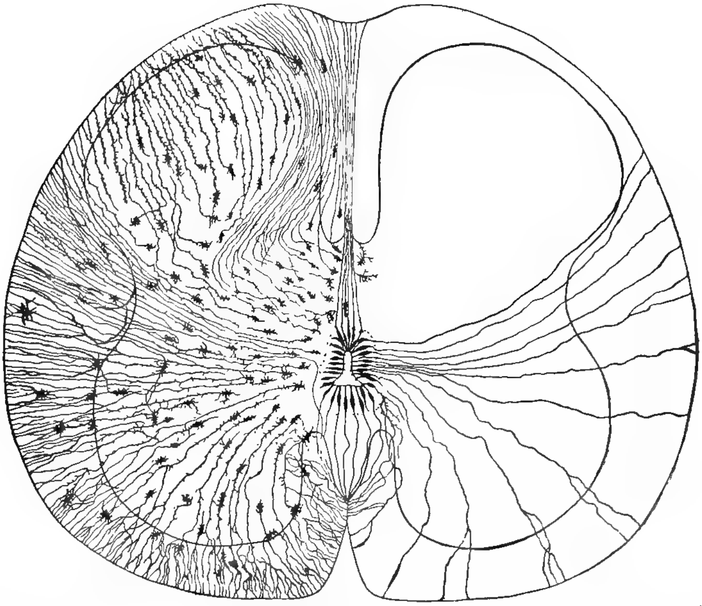
Development of Motor Nerve Roots.—The neuroblast attempting to pass outward between the reticulations of the myelospongium is stopped by the velum confine or external network, and its process is turned aside inward or ventrally; but eventually it succeeds in piercing the external layer, then to become the rudiment of an anterior or motor spinal nerve, which soon gets close to the rudiments of the posterior or sensory roots which have been first formed. The neuroblast remains within, and becomes a true nerve cell. It is thus seen that the nerve fibre and the nerve cell are embryologically and histologically the same unit. The cell nuclei of the columnar cells are arranged in rows, but their ends reach to the surface of the medullary plate. When the closed neural tube or canal is formed, the axes of these cells become disposed round it in a radial manner, as shown in Fig. 36.

FIG. 35.



Neuroblasts from the spinal cord of a five-day chick: *a*, pyriform cell; *b*, cell with first dendrite directed centrally; *c*, cell showing development of the dendrites; *d*, growth cone of a motor fibre. (After Lenhossék.)

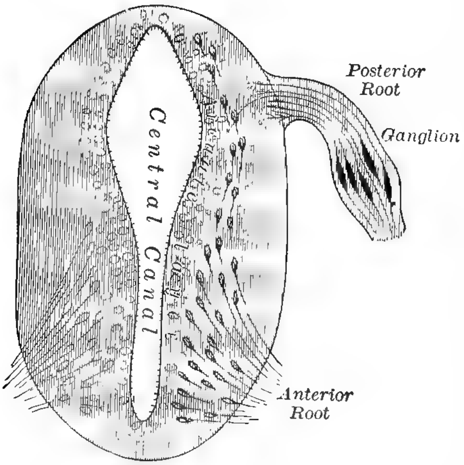
FIG. 36.



The spongioblastic framework of the spinal cord. Cord of a human embryo, fourteen centimetres long, impregnated by the Golgi method. (Lenhossék.)

Development of Sensory Nerve Roots.—The sensory nerve roots do not arise in the central nervous system, but in the posterior spinal ganglia which in the spinal region lie along the courses of the spinal nerves and are present also in some instances along the cranial nerves. When the medullary groove closes to become the neural canal, just at the seam appears an epithelial band or ridge, known as the *neural crest*, which becomes segmented, moves outward, and forms the spinal ganglia. The cells of these ganglia are bipolar, their prolongations being opposite, and passing as fibres, one towards the spinal cord and the other towards the periphery. For these cells, which are designated by Lenhossék as *ganglioblasts*, Baker has proposed the name *œsthesioblasts*. The roots of all sensory spinal nerves and sensory

FIG. 37.



The formation of nerve roots. Section through spinal cord of a human embryo. (Edinger, after His.)

cranial nerves grow from these ganglia into the central organs. The method of the formation of both motor and sensory nerve roots is shown in Fig. 37. The size of the posterior spinal ganglia is in the main proportionate to that of the nerves upon which they are formed. Most of these ganglia occupy the intervertebral foramens externally to the points where the nerves pass through the dura; but the ganglia upon the first and second cervical nerves are exceptions, being placed in the arches of the vertebræ over which the nerves pass; while the ganglia of the sacral nerves are placed within the spinal canal, and the ganglion on the coccygeal nerve in the canal about the middle of the posterior root. Each ganglion gives origin internally to a posterior root and receives an anterior root, the two portions being united into a single mass externally. Careful examinations made on adults show that about as many fibres pass through the posterior root to the spinal cord as have entered the ganglia from the nerves, and therefore it would seem as though a single cell were intercalated in the course of each fibre. A few sensory fibres probably originate in the spinal cord. That practically all the sensory nerves originate in the cells of the spinal ganglia has been settled by the experiments of Waller, Joseph, and others. If a sensory nerve is divided just in front of the spinal ganglion, all its fibres degenerate, the ganglion itself and the root originating in it remaining normal; but if the posterior root is cut just back of the ganglion, only a few fibres of the sensory nerve degenerate. The nerve must, therefore, consist of fibres which originate in the spinal ganglion cells, for the connection between these is unbroken, while the fibres which are destroyed must have come from the spinal cord itself, for they are severed from the latter only and not from the ganglion. Experiments have shown that the cell processes pass out of the ganglion in two directions, and that the fibres which originate in the spinal cord alone pass through it (Edinger).

Development of Lateral Nerve Roots.—We commonly speak of only two sets of nerve roots—Sir Charles Bell's anterior or motor and posterior or sensory; but Bell really divided the nerve roots throughout the central nervous system into three sets, the third being a lateral or respiratory set and containing nerves which excite motions which depend on or are related to the act of respiration. Gaskell says that physiologists have failed to follow this, because the triple arrangement of these nerve roots was not immediately evident, like the separation into the anterior and posterior roots. Every segment of the spinal cord, according to Gaskell, gives origin to two series of roots, a somatic and a splanchnic—the latter being the roots of the nerves supplied to the viscera and bloodvessels; the former of the nerves to the muscles and skin, and to the tissues which are not vascular and visceral. The somatic roots are, in other words, the motor and sensory roots, and in the main are connected re-

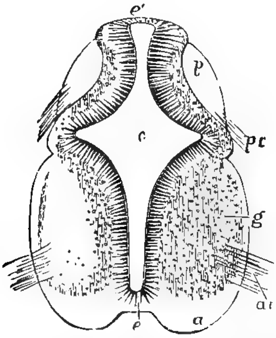
spectively with the cells of the anterior horns and of the posterior spinal regions—ganglia and horns. The lateral roots arise from two columns of nerve cells, namely, the column of Clarke and a column of the lateral horn. The splanchnic roots arising from the column of Clarke become gangliated, their nerves going to vessels, viscera, and glands chiefly, and those arising from the lateral horn are non-gangliated, their nerves going mainly to muscles. These lateral roots are found in greatest number in the region from the second thoracic to the first lumbar segment. The distinction into a ventral or somatic and a lateral or splanchnic group of fibres is, according to His, well marked in the embryo.

All Ganglia Neuraxial Outgrowths.—All the ganglia lying outside of the great neural canal are best regarded as encephalospinal offshoots and outgrowths; and from this point of view it is certainly best to include the posterior spinal and intracranial ganglia in the same system which includes the lateral and collateral chains, and the cardiac, vascular, and other distal ganglia. Every fibre of the gangliated system before it reaches its final destination fuses more or less with other fibres from the neuraxis. The Gasserian ganglion and other intracranial ganglia, as well as the posterior spinal ganglia, have developed as offshoots of the encephalospinal nerves or nerve roots close to their central terminations. Even some of the cranial nerves which are recognized as not now having ganglia have probably at one time had at least traces of these structures. Motor nerves in the adult are wanting in such ganglia, the groups of great cells in the cord and oblongata being sufficient for their innervation, and yet some nerves which are recognized as almost purely motor show traces of sensory ganglia. Thomsen has found even in the adult traces of preexistent ganglionic structure in the root of the third nerve, and Gaskell finds similar traces in the roots of the fourth, the seventh, and the motor roots of the fifth; these indicate the preexistence of sensory elements in the root, and it is therefore probable that these nerves and ganglia have all been originally developed as outgrowths from the neural crest.

Development of the Spinal Cord.—A transection of the cervical cord of a human embryo of six weeks, after Koelliker, is seen in Fig. 38, and shows the manner in which the horns and columns of the cord first appear. At about this time can be seen the beginning of the anterior white column, and to a less extent the posterior, with their roots, the former on the ventral and the latter on the dorsal aspect of the neural canal. Only a few neuroblasts representing gray matter separate at this stage the posterior white columns from the neurogliar lining of the canal, but a little later the gray horns grow outward, and the anterior and posterior fissures appear. The cord changes somewhat in shape from stage to stage in its development. The afferent or sensory nerve fibres become united with the

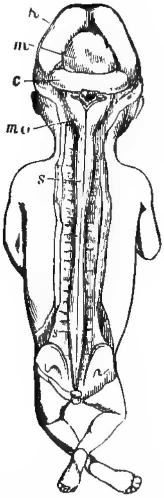
dorsolateral portion, and the efferent or motor take origin from the ventrolateral. Lateral nerve roots also appear. The cylindrical form of the spinal cord is assumed after the development of the

FIG. 38.



Transection of the cervical spinal cord of a human embryo of six weeks: *c*, central canal; *e*, its epithelial lining; *e'* (superiorly), the original place of closure of the canal; *a*, ventral or anterior columns; *g*, gray substance of anterolateral horn; *p*, dorsal or posterior column; *ar*, ventral or anterior roots; *pr*, dorsal or posterior roots. (Quain's Anatomy, from Koelliker.)

FIG. 39.



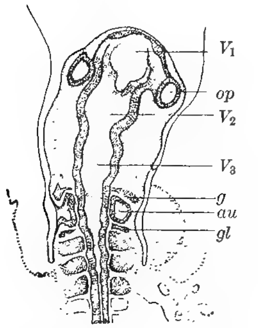
Brain and spinal cord exposed from behind in a fetus of three months: *h*, the hemispheres; *m*, the mesencephalic vesicle (quadrigeminal body); *c*, the cerebellum; below this are the oblongata (*mo*) and fourth ventricle, with remains of the membrana obturatoria. The spinal cord (*s*) extends to the lower end of the sacral canal, and shows brachial and crural enlargements. (Quain's Anatomy, from Koelliker.)

lateral columns. At first the cord and its canal are of nearly equal length (Fig. 39); but after about the fourth month the vertebral column begins to grow more rapidly than the cord, giving eventually the well known relations of the spinal cord and its nerves to the canal, as seen in Fig. 7, page 6.

The Cerebral Vesicles.—After the neural groove has reached a certain stage of development its cephalic or anterior end enlarges, bends downward, and shows three dilatations which are called primary cerebral vesicles. Soon the anterior and posterior vesicles each subdivide into two, one at each side; the middle remains single. These five vesicles give rise to the five rudimentary divisions of the brain—the forebrain, hindbrain, midbrain, interbrain, and afterbrain. The rudiments of the optic nerve and retina grow from the sides of the forebrain. In Fig. 40 these vesicles as in an embryo chick thirty-eight hours old, and also some structures connected with the cranial nerves, are seen. Fig. 41 is a diagrammatic representation, modified from Mihalkovics and Edinger, of these five cerebral vesicles. The figure also shows a fold of the interbrain which afterwards becomes the conarium or pineal body; a depression of its roof in the position of the posterior commissure; and a pushing upward of the epithelium of the pharynx, which is the rudiment of a portion of

the hypophysis or pituitary body. From time to time flexures occur at various places, the general trend being forward and downward: the first flexure is opposite the base of the midbrain; the

Fig. 40.



Head of embryo chick of thirty-eight hours, viewed from above: V_1, first cerebral vesicle—third ventricle; *op*, optic vesicle; V_2, second cerebral vesicle; V_3, third cerebral vesicle = fourth ventricle; *g*, facial ganglion; *au*, auditory vesicle; *gl*, glossopharyngeal ganglion. Enlarged about twenty times. (After Duval.)

Fig. 41.

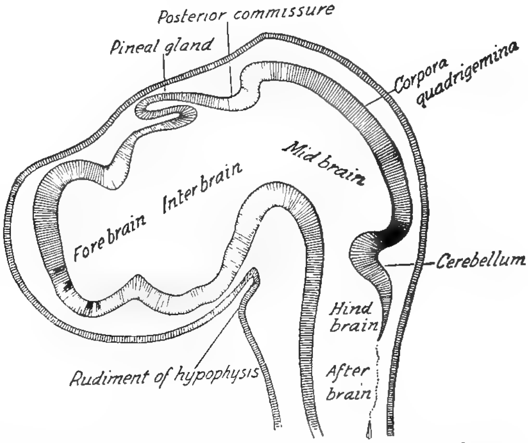


Diagram of the five brain vesicles indicating the subsequent great subdivisions of the brain. (After Edinger-Mihalkovics.)

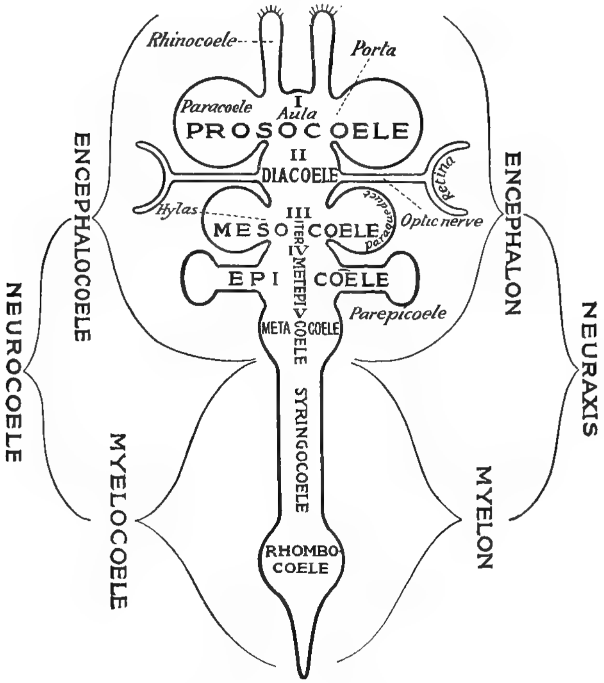
second in the region of the hindbrain or pons, in this case the concavity being directed dorsally; and a third ventrad at the junction of the hindbrain with the spinal cord. The brain thus gradually assumes curious crooks and twists, developmental additions constantly taking place.

The Neuraxial Cavities.—The cavities of the neuraxis, the relics of the neural groove and cerebral vesicles, may be regarded as parts of one great whole, for which the term *cœle* or *cœlia* has been proposed by Wilder, who by appropriate prefixes has also correlated the names of the several divisions of the cœlia with one another, and with the special segments of the nervous system. These cavities are the so-called ventricles of the brain, with their horns, their connecting foramens and canals, and the central spinal canal, although the last is usually so reduced in size in the adult as to be scarcely recognizable as a cavity. Figure 42 has been combined and slightly modified from two diagrams by Wilder, composed by him to express his morphological ideas respecting the segmentation of the brain and the continuity of all the neuraxial cavities.[1] The cephalic or anterior boundary of the cavities is the

[1] In a letter to the author, dated March 18, 1894, Prof. Wilder calls attention to the note on p. 114 of the Reference Handbook of the Medical Sciences, Vol. VIII.,—the note referring to whether or not the olfactory bulbs, their crura, part of the precommissure, and a corresponding portion of the aula, should be regarded as a sixth segment,—and adds, "Certain facts of development and

terma or lamina cinerea, the thin layer of gray matter immediately in front of the recess formed by the chiasm. The *prosocœle* represents the cavity of the prosencephalon or forebrain; the *paracœles*, its lateral extensions, corresponding to the lateral ventricles; while the *rhinocœle*, an imperfectly developed extension of the latter, corresponds to the olfactory bulb. The third ventricle of the brain, as

FIG. 42.



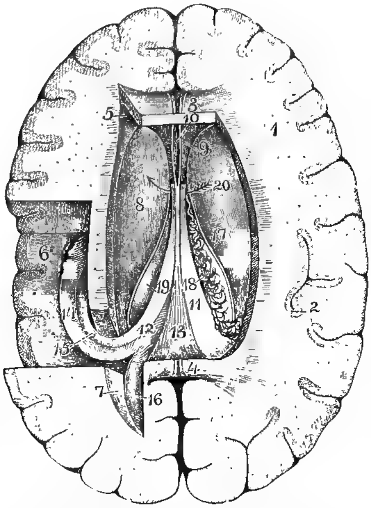
Scheme of the cavities of the brain (and five encephalic vesicles), and of the myelic cavity or central spinal canal. (After Wilder.)

usually described, is subdivided in this diagram into two segments, a larger, or *diacœle*, between the thalami; and a smaller, the *aula* or hall, which constitutes the middle portion of the prosocœle, with which it connects at either side by the *porta* or foramen of Monro. The optic nerve and retina are represented as lateral extensions of the diacœle, because they are originally hollow outgrowths of the diencephalic vesicle prior to the formation of the second cerebral vesicle. The *mesocœle* corresponds to the iter or Sylvian aqueduct, which is also represented with lateral extensions (*hylas* and *paraque-*

comparative anatomy indicate that the olfactory portion of the brain is primary, and morphologically principal, the cerebral portion secondary and subordinate. Probably there should be recognized a segment, the *rhinencephal*, not in the Owenian sense, but as composed of one pair of olfactory bulbs and their cavities united across the meson by part of the precommissure, and part of the aula."

ducts), some adult human brains presenting a slight groove at each side of this aqueduct, and the mesencephalic cavity of frogs, some reptiles, and all birds being distinctly in three parts. Wilder preferably subdivides the cavity of the fourth ventricle into two parts, the *epicœle* and the *metacœle*, corresponding respectively to the hindbrain and the afterbrain; but it is usually more convenient for descriptive purposes to use the term *metepicœle*, which includes both epicœle and metacœle, as synonymous with fourth ventricle. The correlations of these cavities to the five cerebral vesicles are as follows: prosocœle to the first; diacœle to the second; mesocœle to the third; epicœle to the fourth; and metacœle to the fifth. The anatomical textbooks usually speak of a fifth ventricle, a cleft in the septum between the lateral ventricles or paracœles; but this is not a true brain cavity, and is properly designated by Wilder as the *pseudocœle*, or false ventricle. As the brain develops, this appears first as a part of the great longitudinal fissure, from which in man it subsequently becomes isolated by the formation of the callosum. Each of its sides consists of an outer layer of white matter continuous with the callosum, and a thinner internal lamina of gray substance. The central spinal canal, or *myelocœle*, is divided into its main length, or *syringocœle*, and a lower dilated portion, the *rhombocœle*, which is usually called the inferior rhomboidal sinus or terminal ventricle. This method of representing the neuraxial cavities gives a bird's eye view of the brain as a series of mesal segments, and shows the relations of these to the cavity of the spinal cord.

Fig. 43.



Horizontal section of the cerebral hemispheres, the callosum removed, and the lateral ventricles exposed: 1, white substance of the interior of the cerebral hemispheres; 2, gray cortex of the convolutions; 3, 4, extremities of the callosum; 5, precornu (anterior horn) of the left lateral ventricle or paracœle; 6, medicornu (middle or descending horn); 7, postcornu (posterior horn); 8, striatum; 9, septum; 10, pseudocœle (fifth ventricle); 11, fornix; 12, posterior crus of the fornix; 13, attachment of the fornix to the under part of the callosum; 14, hippocamp; 15, fimbria; 16, calcar; 17, tenia (tænia semicircularis); 18, choroid plexus; 19, edge of the thalamus; 20, arrow through the porta or foramen of Monro. (Leidy.)

The Extensions and Relations of the Paracœles.—In Fig. 43 is represented a horizontal transection of the cerebrum, so made as to expose the paracœles or lateral ventricles, and to show their extensions or horns, and some of their most important related solid structures. The third ventricle or diacœle is not shown, but it is situated between the thalami, and chiefly beneath the fornix and its attachments at 12

and 13. The *fimbria*, one of the parts seen in the figure, is a white band along the inner side of the hippocamp. The tenia or tænia semicircularis is a whitish streak between the caudatum or caudate nucleus and the thalamus. The extensions of the paracœles called the anterior, middle, and posterior horns—or the precornu, medicornu, and postcornu—are shown at 5, 6, and 7; the septum and the pseudocœle, at 9 and 10. The caudatum appears as a gray eminence tapering behind, external to the thalamus, only a small portion of which is seen at 19. The hippocamp and calcar are white eminences or ridges which correspond to the infoldings of two great fissures, the hippocampal and the calcarine. The position and appearance of the fourth ventricle or metepicœle have been indicated in Figs. 3, 5, and 6.

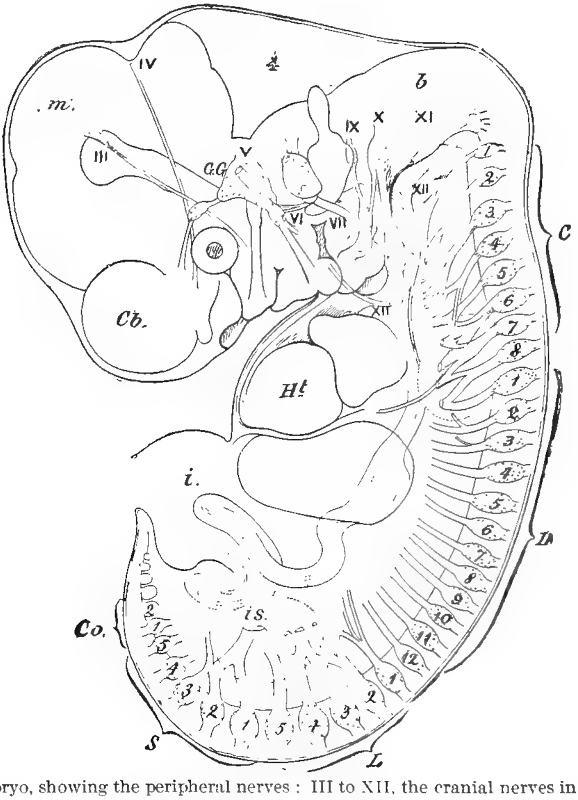
Relationships of Cerebral Vesicles to the Parts of the Fully Developed Brain.—The subjoined table, modified from Quain's Anatomy, shows the relationships which the cerebral vesicles subsequently bear to the subdivisions of the brain, both cavities and solid structures:

I. Anterior primary vesicle or forebrain.	First secondary vesicle (prosencephalon).	Aula (cephalic end of third ventricle); porta (foramen of Monro); paracœles (lateral ventricles); cerebral hemispheres; olfactory bulbs and tracts; striata; callosum; fornix.	
	Second secondary vesicle (diencephalon or thalamencephalon).	Diacœle (third ventricle); optic nerve and retina; thalami; hypophysis; conarium.	
II. Middle primary vesicle or midbrain.	Third secondary vesicle (mesencephalon).	Mesocœle; iter; quadrigeminum; crura.	
III. Posterior primary vesicle or hindbrain.	Fourth secondary vesicle (epencephalon).	Epicœle; cerebellum; pons.	Metepicœle (fourth ventricle).
	Fifth secondary vesicle (metencephalon).	Metacœle; oblongata.	

Segmental Character of the Entire Nervous System.—The central nervous system is to be regarded as a series of segments. The segmental character of the brain has been illustrated in the consideration of the brain vesicles and neural cavities. The spinal cord may be considered as a series of organs vertically linked together—a chain of segments placed one above another. In many lower animals, as in fishes and snakes, this arrangement is obvious, and the spinal cord becomes a series of alternating swellings and constrictions. Each segment may be viewed as a distinct spinal cord for a definite area of the body, namely, that muscular area to which its ventral or anterior roots proceed, and that sensitive area—skin, tendon, muscle, mucous membrane, or viscus—to which the fibres

of the dorsal roots are distributed. According to Hill, the recognition of the segmental succession in the arrangement of the nuclei of the nerves in the spinal cord is due to Aeby. In the Hunterian Lectures, in 1885, Hill applied this principle of segmentation to the brain, or at least to the cranial nerves, attempting to fix the position in the cerebral axis of the nuclei of these nerves by a consideration

Fig. 44.



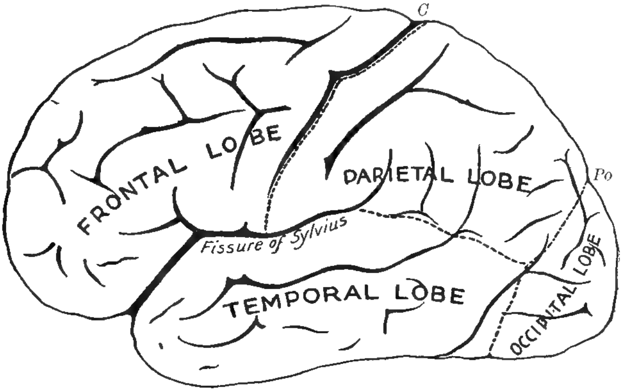
Human embryo, showing the peripheral nerves: III to XII, the cranial nerves in order from the third to the twelfth; Cb, developing cerebral hemispheres; m, midbrain; 4, fourth ventricle; b, commencement of oblongata; C (1 to 8), the cervical nerves and ganglia on their dorsal or posterior roots; D (1 to 12), the thoracic nerves and ganglia on their dorsal roots; L (1 to 5), the lumbar nerves and ganglia on their dorsal roots; S (1 to 5), the sacral nerves and ganglia on their dorsal roots; Co (1 to 2), the coccygeal nerves and ganglia on their dorsal roots; Ht, ventricle of heart; i, intestine; is, sciatic nerve cut at its origin. (His.)

of their segmental distribution in the head. The spinal nerves and the cranial nerves from the third to the twelfth, as observed in the human embryo, are shown in Fig. 44, as are also the relations which these nerves bear to one another, and to the development of the head and of the spinal column.

Lobes of the Cerebrum.—The lobes as usually given are, on the lateral aspect of the cerebrum, the *frontal*, *parietal*, *temporal* or

temporo-sphenoidal, and *occipital*, as shown in Fig. 45. The frontal lobe reaches backward to the central fissure or fissure of Rolando, and below to the horizontal limb of the fissure of Sylvius; the parietal in front to the central fissure, and behind to the parieto-occipital

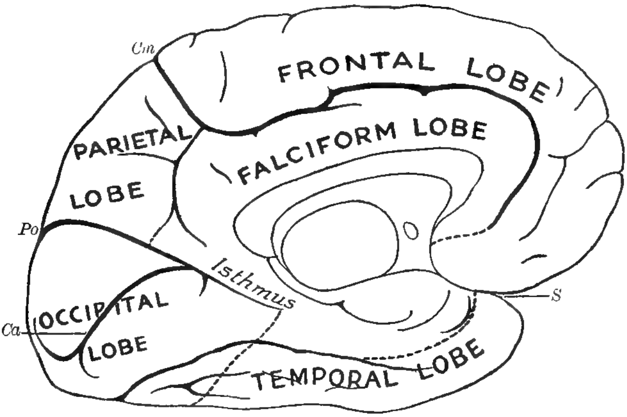
Fig. 45.



Lobes of the cerebrum as usually given by anatomists, lateral aspect: *C*, central fissure or fissure of Rolando; *Po*, parieto-occipital fissure. (Modified from Morris's Anatomy.)

fissure; in front below to the posterior arm of the Sylvian fissure; below and behind it is not usually fully separated from the temporal and occipital lobes by any anatomical landmarks, although it is at times by a short but well defined vertical fissure corresponding to

Fig. 46.



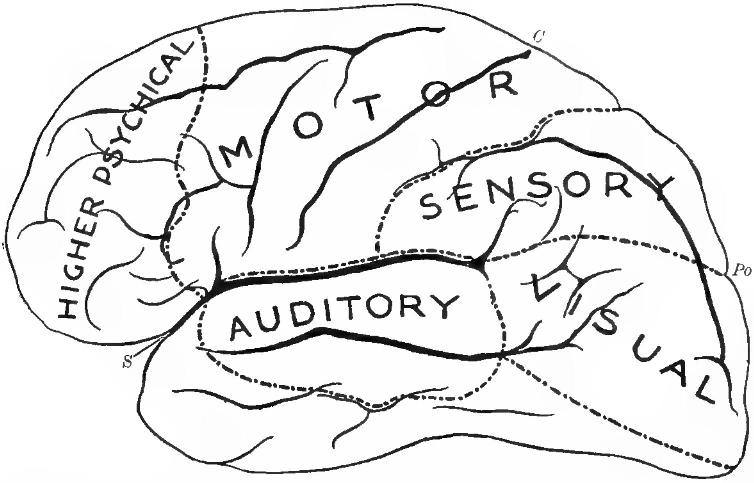
Lobes of the cerebrum, mesal aspect: *S*, Sylvian fissure; *Cm*, calloso-marginal fissure; *Po*, parieto-occipital fissure; *Ca*, calcarine fissure. (Modified from Morris's Anatomy.)

one of the "ape fissures." The boundaries of the temporal and of the occipital lobe are sufficiently indicated by the description of the limitations of the frontal and parietal. On the mesal surface of the hemicerebrum the continuations over the edge of the hemisphere

of the frontal, parietal, and temporal lobes are seen in Fig. 46. A *falciform lobe* has been suggested by Schwalbe; it cannot well be located with any other of the lobes of the brain surface, and embryology and comparative anatomy sustain this lobation. Its outer segment, sometimes spoken of as the *limbic lobe* of Broca, is that shown curving around the callosum; it has an inner segment composed of parts hidden in the view, namely, the septum, the fascia dentata, and a portion of the fornix.

Physiological Lobes.—Instead of the usual anatomical subdivision the brain may be divided, as the result of functional development, into physiological lobes, as shown in Fig. 47 and Fig. 48.

FIG. 47.



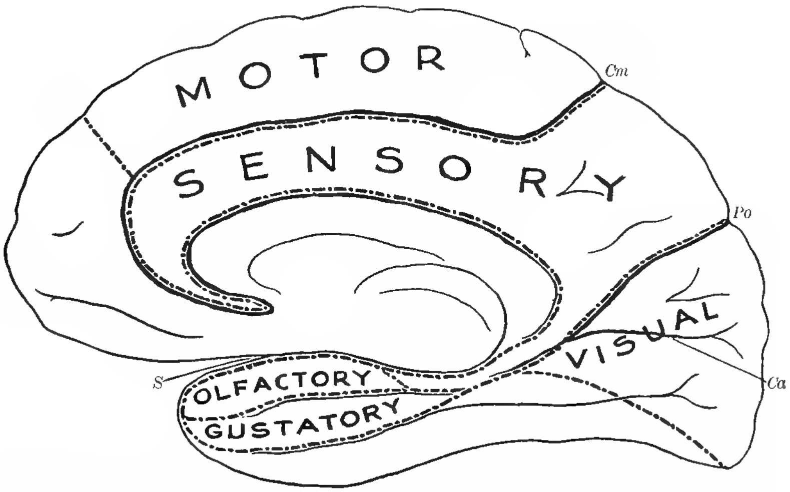
Physiological lobes of the cerebrum, lateral aspect: *S*, Sylvian fissure; *C*, central fissure or fissure of Rolando; *Po*, parieto-occipital fissure.

These lobes may be regarded as (1) a *higher psychical;* (2) a *motor;* (3) a *sensory,* meaning by this a lobe of general or common sensation; (4) a *visual;* (5) an *auditory;* (6) an *olfactory;* and (7) a *gustatory.* The insula, or island of Reil, constitutes a separate lobe of the brain whose functions have not yet been fully determined. According to some authorities, in the middle portion of the temporal lobe, or possibly in the insula, is an *ideational* or *naming* region, where converge the recepts of various kinds as the result of which object and word images arise in the mind. The designation of the prefrontal region as a *higher psychical lobe* is not unobjectionable, and some would oppose altogether the use of the term *psychical* in such a connection; but it perhaps expresses as well as any other the physiological views which have the most support. It is not intended to indicate that it is the only portion of the brain which is psychical; but only that it is related to the highest mental processes, its lesions causing, when sufficiently extensive, a mental deterioration

which is essentially or mainly a defect of the faculty of attention. It is Hughlings Jackson's third or highest level of the nervous system, the acme of human evolution, containing centres of re-representation of the most complex movements of all parts of the body. It might perhaps be designated as the lobe of re-representation or of complex coordinations. The physiological lobes are in the main well demarcated by prominent fissures.

Lobation of the Cerebrum during Development.—Hill has shown that the brain during its growth exhibits a well marked tendency to bulge into lobes ; and he believes that these have a distinct morphological and also a distinct physiological significance. Fig. 49 shows this lobation of the cerebrum. The cephalic end of the cerebrum has the appearance of greatest stability. The appearance of the Sylvian fossa is owing to an intimate relation which exists between the lenticula and the overlying cortex, whereby the island of Reil is fixed and prevented from taking part in the free growth of the rest of the hemisphere. The general surface bulges over the fossa, and soon that portion of the brain which is known as the operculum or Rolandic lobe (*R*) becomes and remains the most distinct of all the lobes of the brain. The ventral and caudal part of the hemisphere bulges forward as the temporal lobe ; while the prolongation backward of its caudal and dorsal portion is obvious in many animals. These prominent *frontal*, *opercular*, *occipital*, and *temporal* bulgings include others which are less obvious. Hill is unable to assign to any of the lobes mentioned, that portion of the surface which lies behind the Rolandic and temporal and in front of the

FIG. 48.



Physiological lobes of the cerebrum, mesal aspect : *S*, Sylvian fissure ; *Cm*, calloso-marginal fissure ; *Po*, parieto-occipital fissure ; *Ca*, calcarine fissure.

occipital lobe, although he finds sufficient indications of the existence upon it of other less pronounced swellings. In the main this method of lobation shown during fetal life corresponds to the physiological lobes as given in Fig. 47.

FIG. 49.

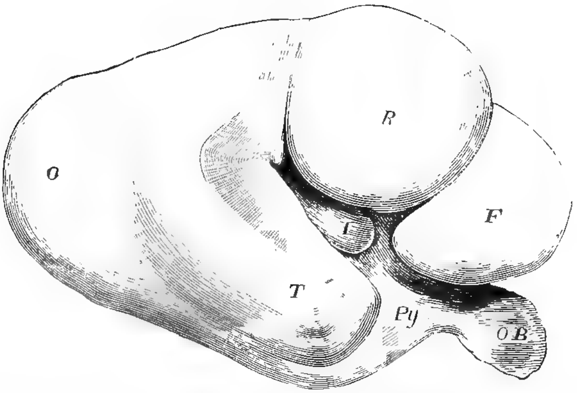
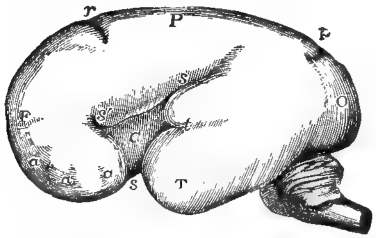


Diagram showing the lobation of the cerebrum: *F*, frontal lobe; *R*, central or Rolandic lobe; *O*, occipital lobe; *T*, temporal lobe; *I*, insula (island of Reil); *Py*, pyriform lobe (uncinate gyre); *OB*, olfactory bulb. (Hill, in the translation of Obersteiner's Anatomy.)

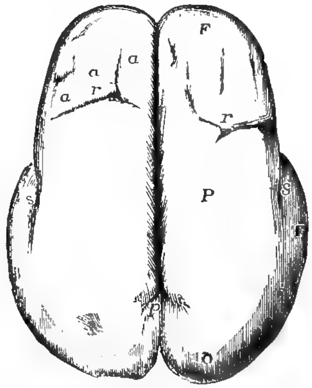
First Appearance of Fissures and Convolutions.—At first the surface of the brain is perfectly smooth, but soon, the enlargement of the cranium not keeping pace with that of the brain, the latter

FIG. 50.



Surface of the fetal brain at six months, viewed from the left side, showing the formation of the principal fissures: F, frontal lobe; P, parietal; O, occipital; T, temporal; *a*, *a*, *a*, slight appearance of sulci in the frontal lobe; *s*, Sylvian fissure; *s′*, its anterior division; within it C, the central lobe; *r*, Rolandic sulcus; *p*, parieto-occipital fissure. (Quain's Anatomy, after R. Wagner.)

FIG. 51.

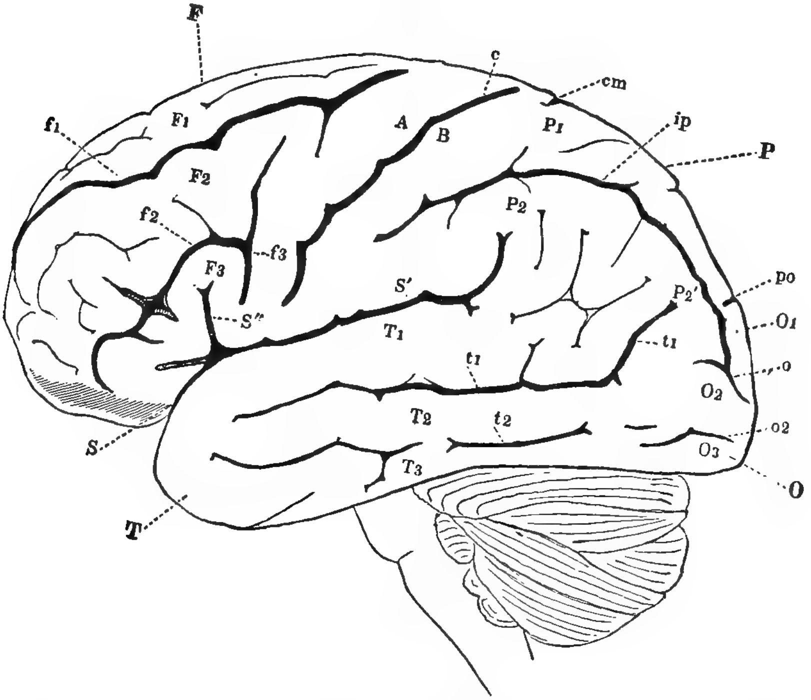


Surface of the fetal brain at six months, viewed from above: F, frontal lobe; P, parietal; O, occipital; T, temporal; *a*, *a*, *a*, slight appearance of sulci in the frontal lobe; *s*, Sylvian fissure; *r*, Rolandic sulcus; *p*, parieto-occipital fissure. (Quain's Anatomy, after R. Wagner.)

begins to be thrown into folds and furrows. The primary infoldings are mostly transverse, although one or two on the mesal surface are parallel with the brain axis. These temporary or primitive sulci, with the exception of three, disappear during the fourth month.

The permanent primitive sulci are, the hippocampal, corresponding with the projection of the cornu ammonis or hippocamp; the parieto-occipital (occipital of Wilder), corresponding with the bend of the postcornu; the calcarine, corresponding with the projection of the calcar; and the Sylvian fissure, corresponding with the curve of the lateral ventricle. The formation of the principal fissures in the fetal brain is shown in Figs. 50 and 51. According to Jelgersma and Cunningham, convolutions form on account of the tendency of the superficial layer or cortex to increase by surface extension, and because of the effort at accommodation as regards space between the gray substance and the white conducting paths; but many interesting theories have been advanced with reference to the origination and multiplication of cerebral fissures and convolutions which cannot be considered in a general textbook on nervous diseases.

FIG. 52.

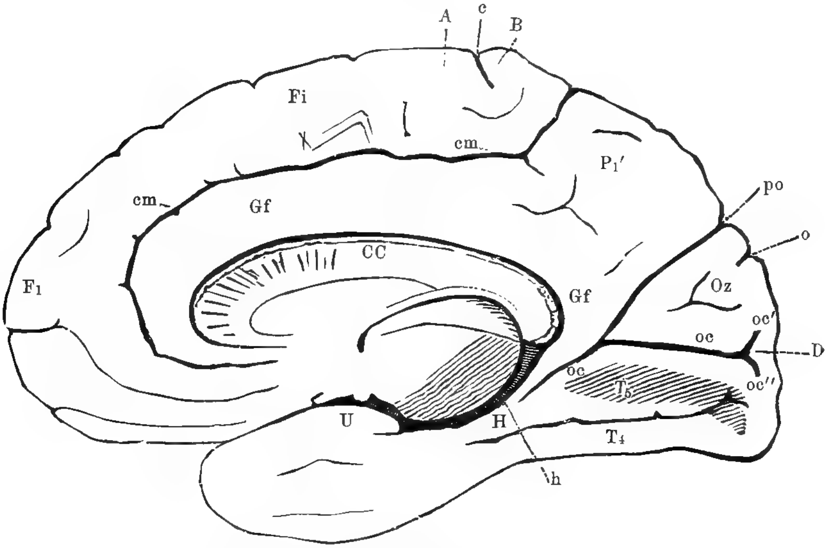


Lateral view of the brain: F, frontal lobe; P, parietal lobe; T, temporal lobe; S, fissure of Sylvius; S′, horizontal, S″, ascending ramus of the same; c, sulcus centralis; A, anterior, B, posterior central convolution; F_1, superior, F_2, middle, F_3, inferior frontal convolution; f_1, superior, f_2, inferior frontal sulcus; f_3, precentral sulcus; P_1, superior parietal lobule; P_2, inferior parietal lobule, including P_2, supramarginal gyrus; P_2', angular gyrus; ip, interparietal sulcus; cm, termination of the calloso-marginal fissure; O_1, first, O_2, second, O_3, third occipital convolution; po, parieto-occipital fissure; o, transverse occipital sulcus; o_2, inferior longitudinal occipital sulcus; T_1, first, T_2, second, T_3, third temporal convolution; t_1, first, t_2, second temporal fissure. (Ecker.)

Fissures and Gyres of the Brain after Full Development.—Ecker's arrangement and nomenclature of fissures and gyres have

been chiefly followed since the appearance in 1869 of his classical work on the convolutions of the human brain; and his well known diagrams of the different surfaces of the brain are given in Figs. 52, 53, 54, and 55. The legends beneath these illustrations are sufficiently descriptive for present purposes. Every student and practitioner should familiarize himself with the position, extensions, and relations of fissures and convolutions, and the most frequent irregularities in fissural and gyral anatomy. It is not always possible to follow a fixed routine in examining the brain; but, when feasible, some general system, like that recommended by Ecker, should be

FIG. 53.

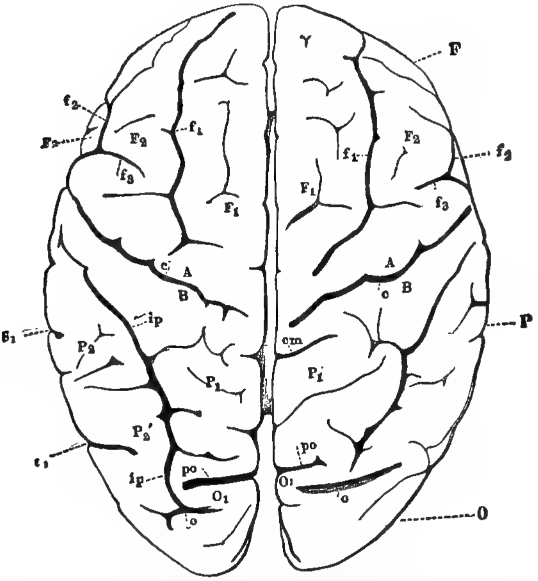


View of the right hemisphere on the mesal aspect: CC, callosum, longitudinally divided; Gf, gyrus fornicatus; H, hippocampal gyrus; h, hippocampal sulcus; U, uncinate gyrus; cm, calloso-marginal sulcus; F, median aspect of first frontal convolution; c, terminal portion of the central sulcus; A, anterior, B, posterior central convolution; P', præcuneus; Oz, cuneus; po, parieto-occipital fissure; oc, calcarine fissure; oc', superior, oc'', inferior ramus of the same; D, gyrus descendens; T_4, lateral occipito-temporal gyrus (lobulus fusiformis); T_5, median occipito-temporal gyrus (lobulus lingualis). (Ecker.)

pursued, proceeding from the more to the less important fissures and gyres. To one acquainted with the brain from books alone the convoluted surface before him may seem at first a tantalizing labyrinth; he may err in expecting that regularity which is seen only in schemes and diagrams. Convolutions change more or less after birth; no two adult brains have exactly the same surfaces. Secondary and tertiary fissures and gyres vary greatly, and even the primary fissures often present exceptional appearances. The great Sylvian cleft is easy of recognition, and the central fissure is scarcely ever hard to pick out, being usually a deep unbridged furrow, about three inches in length, running from above downward and a little forward, nearly

midway of the hemisphere, beginning close to the longitudinal fissure and extending nearly to the Sylvian. Not infrequently it passes into the longitudinal fissure, sometimes with and sometimes without bridging fibres below the surface; it rarely runs into the Sylvian fissure, but this peculiarity has been recorded. A fissure may appear to open completely into another, and yet a bridge be revealed by pulling aside overlapping edges. The first frontal and the inter-parietal fissures, which have bold, continuous outlines in Ecker's plates, are often bridged at one or more points, but commonly these broken fissures should be regarded as one. The parieto-occipital,

FIG. 54.



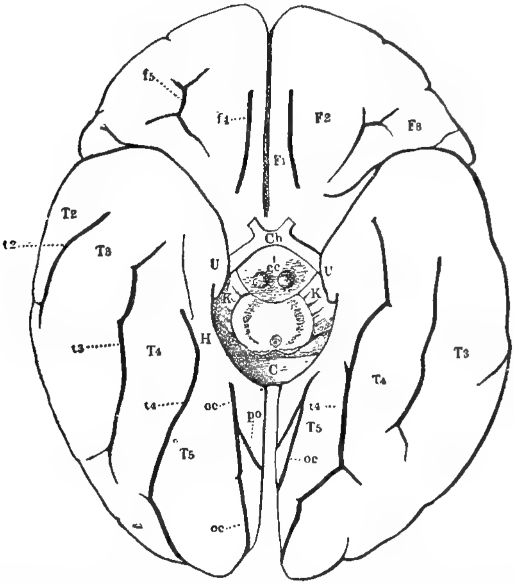
View of the brain from above: F, frontal lobe; P, parietal lobe; O, occipital lobe; S_1, end of the horizontal ramus of the fissure of Sylvius; c, central fissure; A, anterior, B, posterior central convolutions; F_1, superior, F_2, middle, F_3, inferior frontal convolutions; f_1, superior, f_2, inferior frontal sulcus; f_3, precentral sulcus; P_1, superior parietal lobule; P_2, inferior parietal lobule, including P_2, marginal gyrus; P_2', angular gyrus; ip, interparietal fissure; cm, calloso-marginal fissure; po, parieto-occipital fissure; t_1, superior temporal fissure; O_1, first occipital convolution; o, transverse occipital sulcus. (Ecker.)

calcarine, and parallel or first temporal fissures are nearly always deep uninterrupted trenches, the latter usually curving upward in the parietal lobe not far behind the Sylvian fissure.

Fissures of the Human Brain according to Wilder.—As this book is intended as a working manual for students and practitioners, it is considered best to give first the diagrams of Ecker, which are in general use. Other authorities, as Wernicke, Wilder, and Cunningham, have published schemes which differ somewhat from those of Ecker, chiefly in their recognition of certain cerebral markings as fissural integers and in the disjunction of fissures usually regarded

as one. The diagrams of Wilder are given, as representing the most marked deviation from the schemes of Ecker. The main differences are the omission of branches and corrugations; the inclusion of the callosal, inflected, amygdaline, medifrontal, precuneal, postcentral, and subtemporal fissures; the disjunction of the supercallosal from the paracentral, and of the precentral from the subfrontal; the introduction of the exoccipital as probably representing the "ape fissure" of Wernicke; the combination of the transverse occipital of Ecker with the caudal portion of his interparietal as the par-occipital fissure; and the adoption from different writers of fissural

FIG. 55.

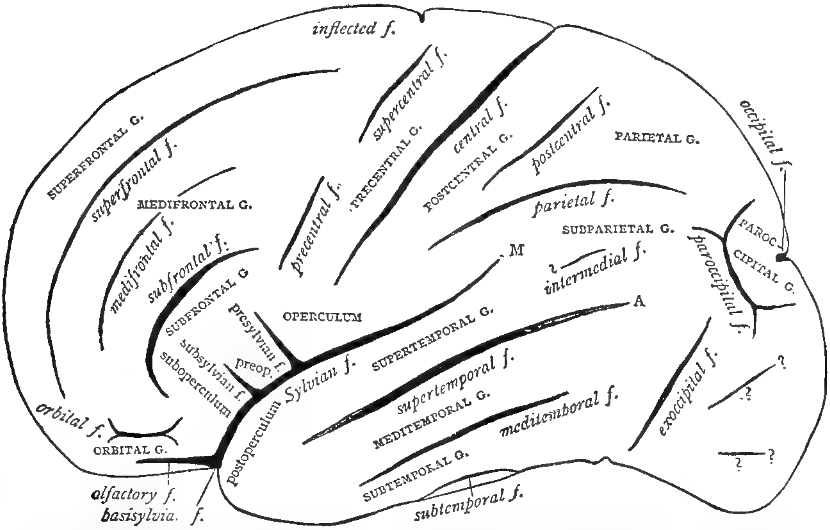


View of the brain from below: F_1, gyrus rectus; F_2, middle, F_3, inferior frontal convolution; f_4, olfactory sulcus; f_5, orbital sulcus; T_2, second or middle, T_3, third or inferior temporal convolution; T_4, lateral occipito-temporal gyrus (lobulus fusiformis); T_5, medial occipito-temporal gyrus (lobulus lingualis); t_4, inferior occipital temporal sulcus; t_3, inferior, t_2, middle temporal fissure; po, parieto-occipital fissure; oc, calcarine fissure; H, hippocampal gyrus; U, uncinate gyrus; Ch, chiasm; cc, albicantia; KK, crura; C, callosum. (Ecker.)

names of a single word each. The outlines and locations of the principal fissures shown in these diagrams are derived from photographs of the cerebrum of a mulatto. The differences in nomenclature will be sufficiently clear from a comparison of the two sets of diagrams.

Variations in Fissural and Gyral Arrangement.—All published schemes of fissures and gyres must be considered more or less imperfect, as they have been based upon investigations of lower animals and of the human brains most frequently accessible, which commonly have been instances of arrested or irregular development—the brains

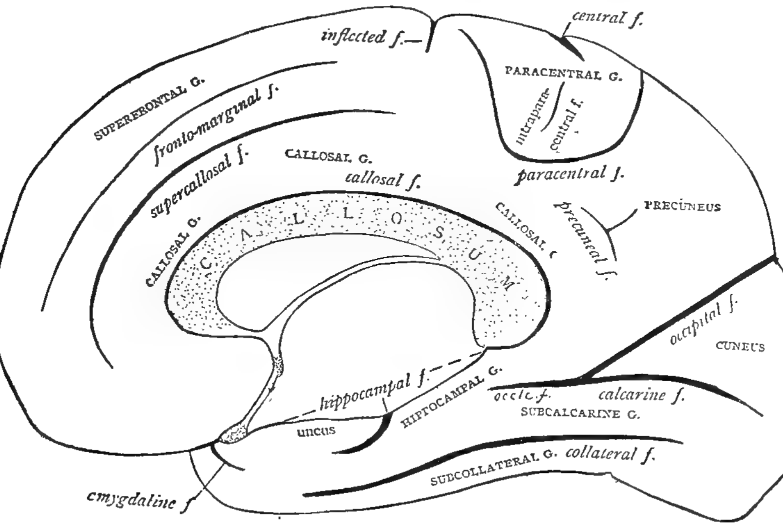
FIG. 56.



Lateral aspect of the left hemicerebrum: A, angular gyrus; M, marginal (or supramarginal) gyrus; *f*, fissure; G, gyrus; preop., preoperculum. The interrogation points near the caudal end indicate Professor Wilder's doubts as to the existence of constant fissures in these places, or as to what they should be called if they do exist. (Wilder.)

of criminals, idiots, paranoiacs, and others belonging to the ignorant, vicious, or insane classes. Comparatively few studies have been made of the brains of the intelligent and educated, although much more is promised, through the formation of societies the members of

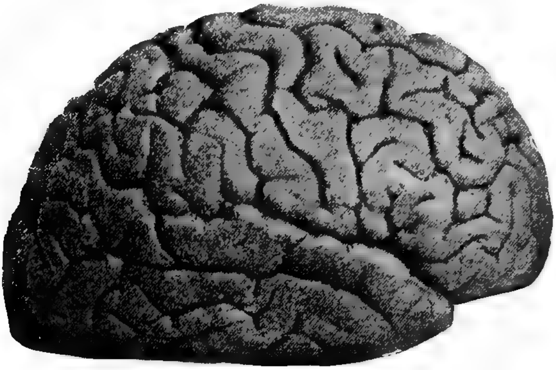
FIG. 57.



Mesal aspect of the right hemicerebrum: G, gyrus; *f*, fissure; *occlc*, *f.*, occalcarine fissure, for occipito-calcarine, the common stem of the fissures demarcating the cuneus. (Wilder.)

which have willed their brains to their associates for scientific purposes. Simplicity of structure, particularly of the frontal fissures and gyres, seems to indicate an inferior order of brain; but such indications are not to be accepted without reserve, as in a few in-

FIG. 58.



Lateral aspect of right hemicerebrum of a Chinaman.

stances the brains of the highly intellectual have shown unusual simplicity of the cerebral surface. Considerable variations from the schemes usually given are to be expected. In Figs. 58 and 59 are shown the lateral and mesal aspects of a Chinese brain, and in

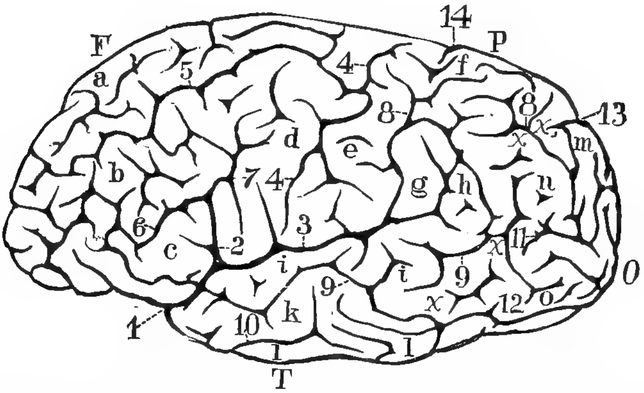
FIG. 59.

Mesal aspect of right hemicerebrum of a Chinaman.

Fig. 60 the left hemicerebrum of a white man showing unusual elaborations and confluences of fissures. The great extent of the supertemporal fissure in the hemisphere shown is a striking peculiarity of the Chinese brain. Figs. 61 and 62 are reproduced from photographs of the right hemicerebrum of a delusional paranoiac of the criminal type. The right hemisphere was shorter and higher than the left, and the whole brain was remarkable for its ape-like and

fetal conditions. A nearly vertical fissure, described in Wilder's diagram as the exoccipital, and termed by Benedikt *Wernicke's fissure,*

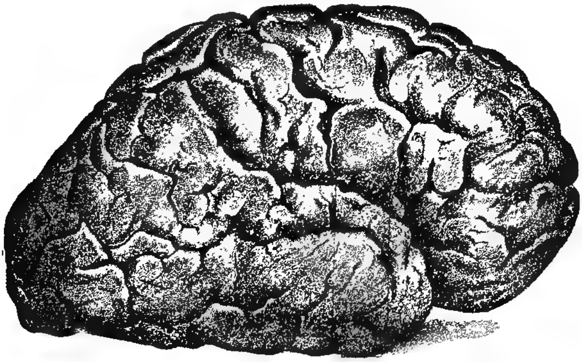
Fig. 60.



Left hemicerebrum of a white man, outer view, one fourth size: F, frontal lobe; P, parietal lobe; O, occipital lobe; T, temporal lobe; 1, Sylvian fissure; 2, anterior branch; 3, posterior branch; 4, central fissure; 5, 6, superior and inferior frontal fissures; 7, precentral fissure; 8, interparietal fissure; 9, 10, superior and inferior temporal fissures; 11, 12, superior and inferior occipital fissures; 13, occipito-parietal fissure; 14, calloso-marginal fissure; *a*, *b*, *c*, superior, middle, and inferior frontal convolutions; *d*, *e*, anterior and posterior central convolutions; *f*, *g*, superior and inferior parietal convolutions; *g*, *h*, angular convolution; *i*, *k*, *l*, superior, middle, and inferior temporal convolutions; *m*, *n*, *o*, superior, middle, and inferior occipital convolutions; *x*, *x*, *x*, *x*, annectant (bridging) convolutions. (Leidy.)

was better defined in this than in any other human brain that I have ever seen, easily demarcating the parietal and temporal lobes from

Fig. 61.

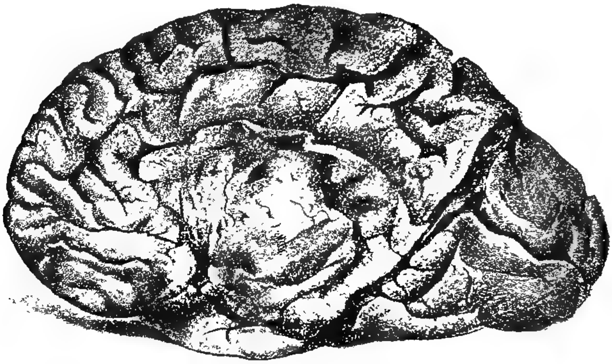


Lateral aspect of right hemicerebrum of a criminal paranoiac.

the occipital upon the lateral aspect of the hemispheres; it corresponded to the inferior portion of the external perpendicular fissure in the ape. These figures illustrating arrested and aberrant development are given, in connection with the diagrams of Ecker

and Wilder, to impress the importance of keeping in mind atypical and irregular fissural and gyral arrangements. By a study of the brains of primates, of fetal brains, and of postnatal brains supposed to be normal, from individuals of different ages, we can obtain a good working knowledge of the average arrangement of the hills and valleys of the cerebral surface. While we have not as yet what might be termed a standard average adult brain, what can be hoped for is that the observer, even the practitioner of medicine of ordinary attainments in cerebral anatomy, shall have a fair idea of brains of different types or degrees of inferiority or superiority of development. Some authorities deny that the fissures and gyres of the human brain can be elucidated by a study of the lower animals, and perhaps in some respects it would be best always to describe and figure what is found without reference to any views as to affinities

FIG. 62.



Mesal aspect of right hemicerebrum of a criminal paranoiac.

with other animals or differences from them; but even so high an authority as Meynert takes as his starting-point the brain of a monkey. Bischoff held that the monkey brain was not a miniature model of the human brain, but represented arrested stages in the development of the latter.

Ental Correlatives of Fissures.—The student should always have in mind the internal elevations which correspond to external fissures—their ental correlatives (Wilder), as such knowledge not only gives a clearer comprehension of the facts of brain development and conformation, but is of direct practical benefit in making autopsies and even in performing surgical operations on the brain. The most striking of these correlations are the calcar to the calcarine fissure, the collateral eminence to the collateral fissure, and the hippocamp to the hippocampal fissure. The caudatum has been suggested as the correlative of the Sylvian fissure, but on grounds not entirely clear.

NOMENCLATURE AND TERMINOLOGY.

General Remarks on Nomenclature and Terminology.—In a textbook on neurology, a section on nomenclature and terminology may at first seem out of place, but anatomical nomenclature is undergoing rapid changes, and this is particularly true of terms used in describing the nervous system. It would be a great gain if general conformity on the part of writers and teachers could be obtained, but, while this is impracticable, the impossibility of bringing about a full reform and of securing a perfect system is not a reasonable excuse for neglecting the subject altogether. Although confusion has occasionally followed attempts at the introduction of new anatomical terms, much practical good has already resulted from the earnest labors of men like Wilder and Gage, whose suggestions as to nomenclature I have to a considerable extent followed, aiming to adopt improved names when this could be done without causing uncertainty or making too much explanation necessary, remembering that the book is for students and general practitioners rather than for anatomists and neurologists.*

Suggestions as to Nomenclature.—Two of the most important suggestions of Wilder, which are gradually becoming more and more appreciated, are the use of appropriate and if possible preexisting mononyms for all parts, and the employment not of heteronyms but of paronyms of these Latin terms formed in accordance with the genius of each language.† He advocates "that, as far as possible, for each part of the central nervous system there be found a name consisting of a single Latin word; that for each such Latin name there be found an English equivalent—not a translation, but a paronym; and that in obtaining these names, Latin and English, due regard be had both to existing nomenclatures and to the establishment of etymological conversion."

Mononyms.—Fig. 63, drawn by Wilder from an actual specimen, represents various parts of the brain as seen in hemisection. It exhibits the manner in which the dura, or outer membrane of the brain, and many of its vessels are related to different parts, showing unusually well the sickle shape of that portion of the dura which is be-

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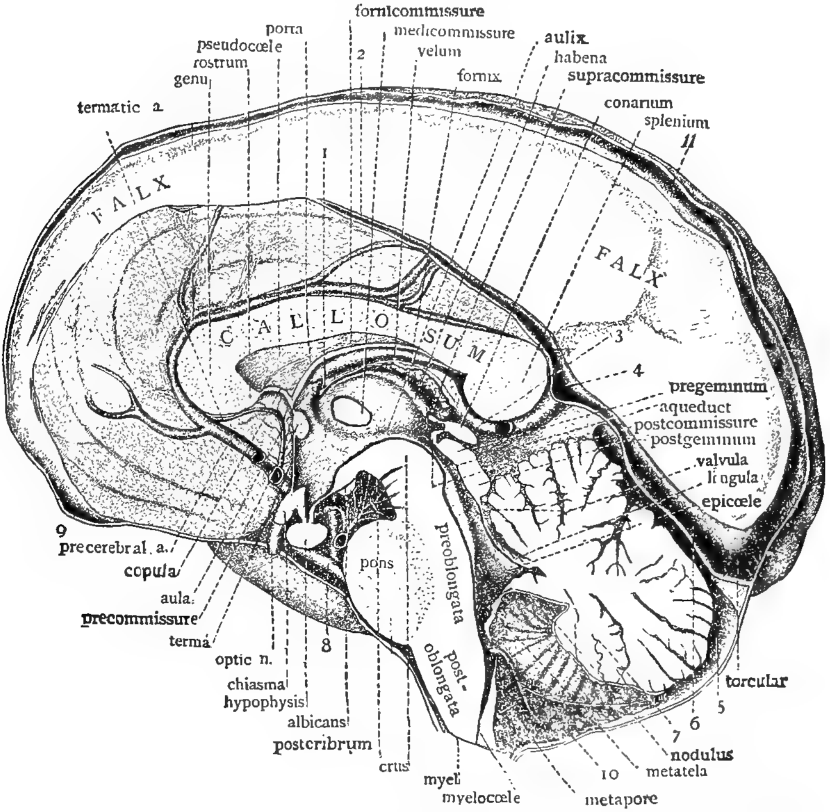
* The material for this section has been drawn chiefly from the writings of Wilder and Gage, and particularly from their contributions to the Reference Handbook of the Medical Sciences, Vols. VIII and IX.

† A *homonym* is a word having the same sound as another, but differing from it in meaning; a *heteronym* is a vernacular term which is a more or less precise translation of a name in Latin or any other language; *paronyms* are words having the same derivation as others to which they are radically allied; they have similar sounds, although sometimes similar and at other times different spellings and meanings. A *mononym* is a term composed of a single word.

tween the two hemicerebrums and is called the *falx*. The numerous descriptive names upon the figure are mononyms, while the common terms for these parts are nearly all polynyms: thus, terma is lamina cinerea; pseudocœle, the fifth ventricle; fornicommissure, the anterior pillars of the fornix; porta, the foramen of Monro; aqueduct, the iter e tertio ad quartum ventriculum; metapore, the foramen of Magendie; postcribrum, the posterior perforated space, etc. The

FIG. 63.



Mesal aspect of the right hemiencephal of an adult: 1, auliplexus; 2, subfalcial sinus; 3, right velar vein; 4, orifice of vein of Galen; 5, falcicula (falx cerebelli); 6, straight sinus; 7, uvula (of cerebellum); 8, tuber cinereum; 9, falx; 10, arachnoid; 11, longitudinal sinus. (Wilder.)

diagram is anatomically very valuable, and reference will be made to it again for purposes of description. In the table which follows illustrations are given of some of the mononyms adopted. In each of these and in others which may be used the evident meaning of the word, or the fact that it is clearly a part of a descriptive term long in use, will usually guide the reader, even if he is not familiar with the changed nomenclature. The mononyms *arbor*, *calcar*, *callosum*, *caudatum*, *lenticula*, *pons*, *dura*, and *pia*, will be at once recognized as

standing respectively for *arbor vitæ, calcar avis, corpus callosum, caudate nucleus, lenticular nucleus, pons Varolii, dura mater*, and *pia mater.* In the table, the Latin mononym, in the nominative singular, is used. In many instances the English vernacular has the same form, and when it has not the change is slight: thus, *diacœlia* and *diacœle; myelon* and *myel; medicommissura* and *medicommissure; postpedunculus* and *postpeduncle; præpedunculus* and *prepeduncle.*

Ala	for	ala cinerea.
Alba	"	white (nervous) matter.
Ansa	"	ansa lenticularis.
Arbor	"	arbor vitæ cerebelli.
Aula	"	cephalic part of third ventricle.
Calcar	"	calcar avis, or hippocampus minor.
Callosum	"	corpus callosum.
Caudatum	"	caudate nucleus.
Cinerea	"	gray matter.
Conarium	"	pineal body.
Crus	"	cerebral crus or cerebral peduncle.
Diacœlia	"	third ventricle less the aula.
Diaplexus	"	plexus of third ventricle.
Diatela	"	membranous roof of third ventricle.
Diencephalon	"	thalamencephalon or interbrain.
Dura	"	dura mater.
Fimbria	"	corpus fimbriatum.
Geniculum	"	geniculate body.
Habena	"	peduncle of the pineal body.
Hippocampus	"	hippocampus major.
Hypophysis	"	pituitary body.
Insula	"	island of Reil.
Lenticula	"	lenticular nucleus, or corpus lentiforme.
Medicommissura	"	middle commissure.
Medipedunculus	"	middle peduncle of cerebellum.
Metaporus	"	foramen of Magendie.
Myelon	"	spinal cord.
Neuraxis	"	cerebrospinal axis.
Oblongata	"	medulla oblongata.
Oliva	"	olivary body.
Pallidum	"	globus pallidus.
Paracœlia	"	lateral ventricle.
Pons	"	pons Varolii.
Porta	"	foramen of Monro.
Postbrachium	"	brachium conjunctivum posterius.
Postcribrum	"	posterior perforated space.
Postpedunculus	"	inferior peduncle of cerebellum.
Præcornu	"	anterior horn of lateral ventricle.
Præpedunculus	"	superior peduncle of cerebellum.
Quadrigeminum	"	corpus quadrigeminum.
Terma	"	lamina terminalis or lamina cinerea.
Thalamus	"	optic thalamus.
Tuber	"	tuber cinereum.
Velum	"	velum interpositum.

Table of Synonyms of Gyres or Convolutions and Lobules.

WILDER.	TURNER.	HUXLEY.	ECKER, CUNNINGHAM, AND OTHERS.
Superfrontal.	Superior frontal.	Supero-frontal.	First or superior frontal.
Medifrontal.	Middle frontal.	Medio-frontal.	Second or middle frontal.
Subfrontal.	Inferior frontal.	Infero-frontal.	Third or inferior frontal.
Precentral.	Ascending frontal.	Antero-parietal.	Anterior central.
Supertemporal.	Superior temporo-sphenoidal.	Antero - temporal.	First or superior temporal.
Meditemporal.	Middle external temporo-sphenoidal.	Medio temporal.	Second or middle temporal.
Subtemporal.	Inferior external temporo-sphenoidal.	Postero - temporal.	Third or inferior temporal.
Subcollateral.	Inferior internal temporo-sphenoidal.		Lateral occipito-temporal or fusiform.
Subcalcarine.	Middle internal temporo-sphenoidal.		Median occipito-temporal or lingual.
Postcentral.	Ascending parietal.	Postero-parietal gyrus.	Posterior central.
Parietal.	Postero-parietal.	Postero-parietal lobule.	Superior parietal.
Subparietal.			Inferior parietal.
Supramarginal.	Supra-marginal.	Angular.	Supramarginal.
Angular.	Angular.		Angular.
Precuneus.	Quadrilateral.	Quadrate.	Precuneus.
Callosal.	Callosal.	Callosal.	Gyrus fornicatus.
Uncus.	Uncinate.	Uncinate.	Uncinate.
Hippocampal.	Hippocampal.		Hippocampal.
Dentate.	Dentate.	Dentate.	Dentate.
Cuneus.	Cuneus.	Internal occipital.	Cuneus.
Paroccipital.	Superior occipital and first external annectant.	Supero-occipital and first external annectant.	First or superior occipital.
Medioccipital.	Middle occipital and second annectant.	Medio-occipital and second annectant.	Second or middle occipital.
Suboccipital.	Inferior occipital, third and fourth annectants.	Infero-occipital and third annectant.	Third or inferior occipital.

The Naming of Fissures and Gyres.—Wilder's names for fissures and gyres are shown in Figs. 56 and 57. As the methods of naming these parts used by different writers vary so much, it may serve a good purpose to give in a table the English gyral synonyms in most general use, to which fissural names closely correspond. The terms most commonly met with are given in the fourth column of the table, although those of Wilder are coming more and more into favor, and anatomists and morphologists are gradually approaching uniformity in their terminology. Only those gyres are tabulated which are most frequently referred to in practical work. Huxley and Turner sometimes use interchangeably "*supero-frontal*" and "*superior frontal*," "*mid-occipital*," "*medio-occipital*," or "*middle occipital*," etc. The *angular* convolution of Huxley does not exactly correspond to the "angular" convolutions of others. Huxley's *postero-parietal convolutions* and *postero-parietal lobule* designate two adjoining but separate regions, and very wisely these terms have been generally discarded. The *supramarginal* and *angular* convolutions together constitute the *subparietal* of Wilder (*inferior parietal* of Ecker). Wilder's *paroccipital* gyre, which practically corresponds to Ecker's first or superior occipital, is named from his paroccipital fissure, which has been given an independent footing because in a large number of brains examined by him it appeared as a distinct fissure. The annectant or bridging gyres connect lobes across great fissures, like the human parieto-occipital, and the perpendicular fissures in simian brains. Their annectant character is more apparent in the brains of apes and other lower animals than in man, although sometimes it is evident in low type human brains, such as the one shown in Figs. 61 and 62. Both the annectant and the adjacent occipital convolutions of Turner and Huxley correspond to Ecker's first, second, and third occipital convolutions. *Operculum*, meaning a "cover" or "lid," is a term applied to a brain region which covers other parts. The *operculum* shown in Wilder's diagram, Fig. 56, includes at least portions of the precentral and subfrontal gyres, and overhangs the insula. The *preoperculum*, *suboperculum*, and *postoperculum* receive their names from their relations to the operculum.

Terms descriptive of the Positions and Relations of Parts.—In describing the positions and relations of parts of the nervous system to each other, and to other structures, it is important to use terms which apply equally to man and the lower animals, as those based upon the normal position of a vertebrate animal, which has six aspects—*cephalic*, *caudal*, *dorsal*, *ventral*, *dextral*, and *sinistral*. Terms derived from these words should be used in preference to common but sometimes ambiguous expressions such as anterior, superior, inferior, external, internal, etc.; although it is difficult for medical writers, students, and practitioners, consistently to disregard them. Cephalic and cephalad are used to designate posi-

tion in or direction towards the head end of the body; caudal and caudad, the same with reference to the tail end of the body; dorsal and dorsad refer to the back, or the part containing the central nervous system; ventral and ventrad, to the belly, or digestive part of the body; while dextral and sinistral are used for right and left, as in everyday life. These terms have much to commend them, and have been largely adopted in this work; their adoption in all cases might prove confusing to the average reader. For the particular uses of cephalic, caudal, ventral, dorsal, etc., as applied to the limbs, the student must be referred to special works or articles, as Gage and Wilder's article in the Reference Handbook of the Medical Sciences; but attention may be called to a few expressions which require to be used with comparative frequency. *Preaxial*, which means situated in front of any transverse axis of the body of an animal, refers to the cephalic or anterior side of the axis of a limb; *postaxial*, to a position behind any transverse axis of the body of an animal, as the caudal or posterior (ulnar or fibular) side of the axis of a vertebrate limb. *Ectal* and *ental* have been introduced to take the place of such terms as external and internal, outward and inward, superficial and profound. They indicate position on or direction towards an aspect or surface farther from an actual or supposed centre than some other part with which it is compared. *Proximal* indicates position or direction near, *distal*, position or direction away from, the attached end of a limb.

Classification of Encephalic Parts.—The table on page 53, which gives, with a few changes, Wilder's provisional classification of encephalic parts according to segments, relations to cavities, and other characteristics, will be found useful not only for gaining familiarity with newly introduced terms, but also as a compact presentation of important embryological and anatomical facts relating to the nervous system.*

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* With a few exceptions the structures described in this table are discussed in more or less detail in the present chapter, so that brief references to a few of the terms will be all that is necessary to make the table clear. The *aulix* is a slight furrow, sometimes called the *sulcus of Monro*, which runs from the porta to the aqueduct on the mesal surface of the thalamus, which it divides into a dorsal and a ventral area, the *habena* being a slight convexity situated at the dorsal margin of this sulcus. The *pala* is a thin structure shaped like a turf cutter connecting the fimbria and tenia in the medicornu. *Tela* is applied to the prolongations of pia found within the encephalic cavities: thus, the *aulatela* is a pial layer which constitutes a portion of the root of the aula, the *diatela* a similar structure for the diacœle, and the *metatela* for the metacœle. *Prosoplexus*, *diaplexus*, *epiplexus*, and *metaplexus* indicate respectively the folds of the pia with their network of vessels found within the prosocœle, diacœle, etc. *Ectocinerea* is descriptive of external or peripheral, and *entocinerea* of internal or central gray matter. The *cappa* is a superficial layer of gray matter, or *ectocinereal lamina* of the quadrigeminal body, situated just beneath the expansion of the optic tracts. *Rimulæ* are the crevices between the cerebellar folia or leaves.

*Encephalic Parts according to Segments, Relations to Cavities, and other Characteristics.**

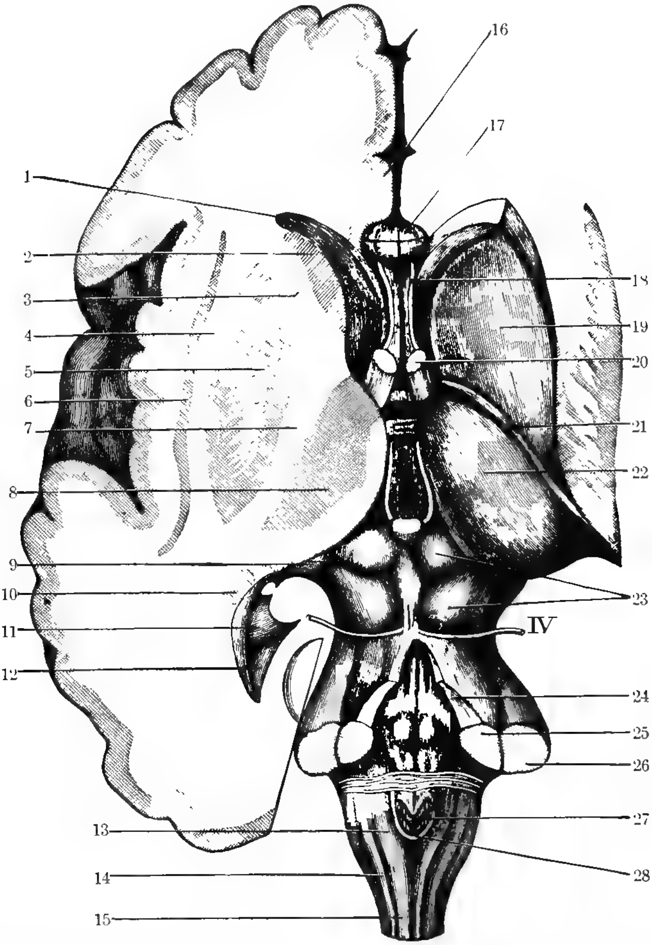
Segments and chief parts.	PROSENCEPHAL: Cerebrum. (Forebrain.)	DIENCEPHAL: Thalami. (Interbrain.)	MESENCEPHAL: Quadrigeminum. (Midbrain.)	EPENCEPHAL: Cerebellum. (Hindbrain.)	METENCEPHAL: Postoblongata. (Afterbrain.)
General cavities.	Prosocœle.	Diacœle.	Mesocœle.	Epicœle.	Metacœle.
Cavitary divisions.	Paracœle, porta, aula.	Aulix, infundibulum.		Lateral recess.	
Floors.	Fornix, caudatum.	Tuber.	Crura.	Preoblongata, pons.	Postoblongata.
Sides.	Hemiseptum, etc.	Thalami.	Quadrigeminum.	Peduncles.	Restis, clava.
Roofs.	Aulatela and callosum.	Diatela.	Quadrigeminum and valvula.	Cerebellum and lingula.	Metatela, obex.
Commissures and decussations.	Callosum, precommissure.	Medicommissure, supracommissure, chiasma.	Postcommissure.	Pons.	Decussation of pyramids; dorsal and ventral commissures.
Marginal parts.	Tenia, fimbria, pala.	Habena.			Obex and ligula.
Telas.	Aulatela.	Diatela.			Metatela.
Plexuses.	Prosoplexus.	Diaplexus.		Epiplexus.	Metaplexus.
Ental elevations.	Caudatum, hippocamp, calcar, collateral, occipital, and callosal eminences.	Habena.			
Ectal elevations.	Gyres, insula.	Pulvinar, pregeniculum, postgeniculum, albicans.	Postgeminum, pregeminum, postbrachium, prebrachium, cimbia.	Folia.	Oliva, clava, tubercle of Rolando.
Ectal depressions.	Fissures.	Trigonum, habenal, fimbrial, and tenial sulci.	Lateral sulcus.	Peduncular sulcus, rimulæ, prepeduncular fossa.	Postfovea.
Ectocinerea.	Cortex.		Cappa.	Cortex.	
Parts of ectocinerea.	Claustrum, amygdala, lenticula.				
Entocinerea.	Caudatum.		Aqueductal cinerea.		
Parts of entocinerea.		Habena ganglion, etc.		Fastigatum, embolus, globulus, dentatum.	
Other parts.		Precribrum, postcribrum.	Intercalatum.	Arbor.	

* Wilder (modified), Reference Handbook of the Medical Sciences, edited by Albert H. Buck, M.D., Vol. VIII, p. 121.

GENERAL ANATOMY.

Important Ganglia, Tracts, and Cavities of the Brain.— In Fig. 64 are shown some of the most important ganglia, tracts,

FIG. 64.

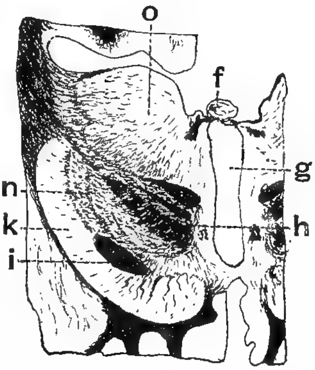


Horizontal section of the cerebrum : 1, precornu (anterior horn); 2, caudatum (caudate nucleus); 3, anterior limb of internal capsule; 4, external capsule; 5, lenticula (lenticular nucleus); 6, claustrum; 7, posterior limb of internal capsule; 8, thalamus; 9, postgeniculum (internal geniculate body); 10, caudatum; 11, hippocampus (hippocampus major); 12, calcar (hippocampus minor); 13, clava; 14, funiculus cuneatus; 15, funiculus gracilis; 16, callosal gyre (gyrus cinguli or gyrus fornicatus); 17, callosum; 18, septum (septum lucidum); 19, caudatum; 20, fornicolumn (anterior pillar of the fornix); 21, tenia (tænia semicircularis); 22, thalamus; 23, gemina (quadrigeminal body); 24, prepeduncle (superior cerebellar peduncle); 25, postpeduncle (inferior cerebellar peduncle); 26, medipeduncle (middle cerebellar peduncle); 27, ala (ala cinerea); 28, obex; IV, fourth or pathetic nerve. (Morris's Anatomy, after Landois and Sterling.)

and cavities of the brain. On the right the appearances are those observed when the lateral ventricles are just uncovered by a horizon-

tal section of the hemicerebrum; on the left, when a section is made through the basal ganglia and adjacent parts. These sections reveal the gyres of the *insula*, the *alba* or white matter of the hemisphere, the *extreme capsule*, *claustrum*, *external capsule*, *lenticula*, *internal capsule*, *caudatum*, *thalamus*, *callosal gyre* or *gyrus fornicatus*, known also as the *gyrus cinguli*, *callosum*, *precornu*, *fornicolumns* or *anterior pillars of the fornix*, *tenia*, *gemina* or *quadrigeminal body*, *postgeniculum* or *internal geniculate body*, *hippocamp*, *calcar*, *ala cinerea*, *obex*, *clava*, *funiculus cuneatus*, and *funiculus gracilis*. In Figs. 65 and 66 are shown vertical transections of the left and of the right hemicerebrum, showing some of the structures above mentioned in different planes, and other important parts, as the *precommissure*, *optic tract*, *tuber cinereum*, *amygdala*, the subdivision of the thalamus into three nuclei, and the nucleus of the *pregeniculum* or *external geniculate body*; also the gyres of portions of the *frontal*, *parietal*, *central*, and *occipital lobes*.

FIG. 65.



Microscopic frontal section showing the basal ganglia and adjacent structures and parts. The section is stained by the method of Weigert-Pal. The fasciculus of Meynert is seen passing downward from the habenula. A little posterior to the plane of this section the third ventricle passes into the aqueduct of Sylvius: o, thalamus; f, pineal body; g, third ventricle; h, internal capsule; i, precommissure; k, putamen; n, pallidum (globus pallidus).

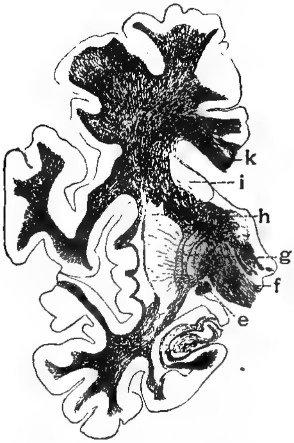
The Striata.—Formerly under the designation corpus striatum, or in the plural corpora striata, two great ganglia in the basal portion of the cerebrum were described as one, the term being given because of the streaked or mixed gray and white appearance, now known to be due to the presence of separate deposits and tracts. The great subdivisions of each striatum are the caudatum, or nucleus caudatus, and the lenticula, or nucleus lenticularis.

The Caudatum.—Each caudatum, caudate nucleus, or intraventricular portion of the striatum, appears, when the lateral ventricles are exposed from above, as a large rounded mass, of pyriform shape, the larger end directed forward so as to project into the frontal lobe, the caudal extremity tapering steadily and passing around the thalamus downward along the roof of the medicornu or descending horn, and then forward again nearly to the cephalic extremity of this horn. This tail-like extension of the caudatum has been described by Dalton as making a *surcingle* to the thalamus. It ends at the amygdala of the temporal lobe. The caudatum and the lenticula usually merge in front for a short distance, and are in close relation below with the cinerea of the precribrum, or anterior perforated space, at the base of the brain.

The Lenticula.—The lenticula, lenticular nucleus, or extraven-

tricular portion of the striatum, does not, like the caudatum and the thalamus, appear upon laying open the paracœles, but is revealed by making deeper sections into the hemispheres. Below it is continuous with the claustrum, as well as with the caudatum and the postcribrum. It is large, extending between the claustrum and both the caudatum and the thalamus for nearly their entire length. In horizontal section it is shaped like a biconvex lens, one curve directed outward and the other inward, the outer having a larger radius than the inner; and in sagittal section it is also approximately lens shaped.

FIG. 66.



Microscopic frontal section somewhat more anteriorly situated than the one represented in Fig. 65. The cornu ammonis, external and extreme capsule, and claustrum are represented in the drawing, but are not designated by letter: e, optic nerve; f, fibres from the internal capsule; g, pallidum; h, putamen; i, caudatum; k, fibres to callosum.

A transection, as in Fig. 65, indicates the interrelations of the lenticula, caudatum, thalamus, and other parts, and also shows that the lenticula is composed of at least three distinct subdivisions separated from each other by thin white layers, which can readily be made out in a fresh brain. The outer larger segment, which is more distinctly separated from the middle than are the middle and inner from each other, is of a dark reddish color, and is called the *putamen*, while the middle and inner segments taken together are known as the *pallidum* (or *globus pallidus*).

The Internal, External, and Extreme Capsules.—The internal capsule is a broad band of white matter to the inner side of the lenticula, and between it and both the caudatum and the thalamus. It is shown in various illustrations, as in Figs. 64, 65, and 66. It presents two limbs, an anterior and a posterior, which are demarcated by a *knee* or bend. The external capsule is a narrower white band between the lenticula and the claustrum. Between the claustrum and the insula is a third band or sheet of alba, usually unnamed, but which has been called the *extreme capsule* or *lamina of the Sylvian fossa*.

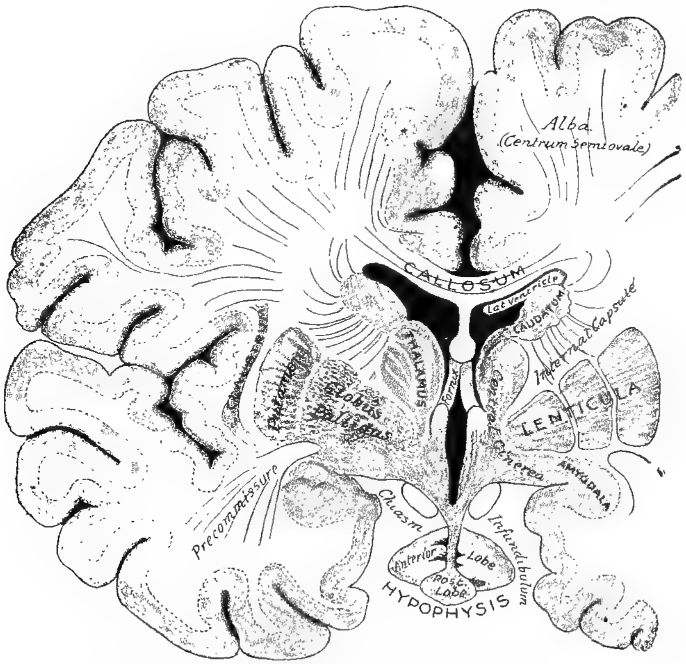
The Claustrum.—The claustrum, shown in Figs. 64, 65, and 66, is a thin sheet of cinerea—an elongated layer or expansion of ganglion cells lying laterally to the external capsule. At its cephalic end it fuses at one point with the amygdala, and it sends small projections outward into the extreme capsule towards the gyres of the insula.

The Amygdala.—The amygdala, shown in Figs. 65 and 67, is a nodular mass of cortical cinerea at the apex of the temporal lobe, just at the termination of the medicornu, into which it bulges as a

tubercle, sometimes called the *amygdaloid tubercle.* It lies ventrally to the lenticula, and is in contact at one place with both the lenticula and the caudatum.

Embryonic Relations of the Claustrum, Caudatum, and Lenticula.—A transection through the forebrain of a human embryo of about ten or twelve weeks shows the rudiments of the striatum projecting upward into the still large forebrain vesicle ; also a rim of cortical matter near the free border of the striate body, and connected at one of its extremities with a narrow layer of the cortex proper. Sections made later show enlargements of these layers of gray matter both in the cortex and in the striatum, and white fibres beginning to separate the latter into two parts, the caudatum and the lenticula. Both, in like manner, are soon separated from the

Fig. 67.

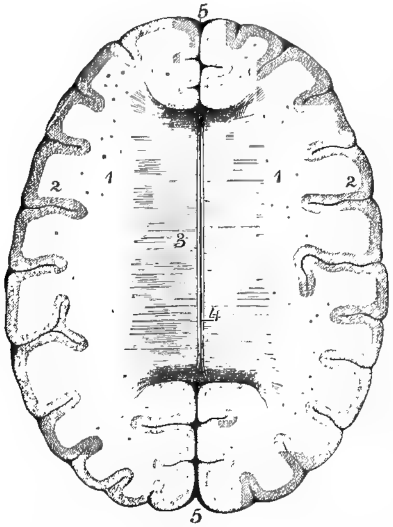


Frontal section through the forebrain. (After Edinger.)

cortex, except at one point at the base of the brain (Edinger). It is thus seen that these great masses of gray matter, which in the adult brain appear to be interior and separate deposits, are in origin related to the cortex, from which they are pushed out in the process of development ; and it is probable that they are cortical in function as in genesis. The student without some knowledge of brain development tends wrongly to regard the thalamus and the striata, because of their juxtaposition, as having functions on the same level

or plane. The claustrum even more evidently than the striata is related genetically to the cortex. The relations of the various parts described are beautifully shown in one of Edinger's diagrams, Fig. 67.

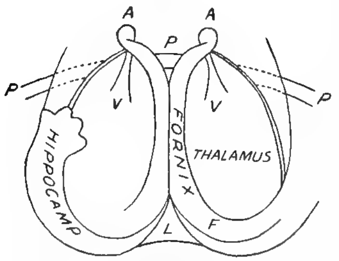
FIG. 68.



Horizontal transection of the hemispheres of the cerebrum on a level with the callosum: 1. white substance of the hemispheres, dotted with divided bloodvessels; 2. gray cortex of the convolutions; 3, callosum, with the direction of its fibres indicated by transverse striæ; 4, longitudinal median striæ; 5, cephalic and caudal portions of the great longitudinal fissure. (Leidy.)

Commissural Structures of the Brain.—The chief structures uniting the hemicerebrums are the *callosum*, *precommissure*, *medicommissure*, and *postcommissure*. The fornix is sometimes spoken of as a commissural structure, but it is rather, on each side, a great tract, the hemifornix, the bodies of the hemifornices coming together in the median line. The distinction between a true commissure, or part uniting related portions of the two sides of the neuraxis, and a decussation, or tract crossing the median line and joining different regions or levels, should be remembered. According to some authorities, the postcommissure is not a true commissure, but in part at least a decussation of the fibres of the fillet. The precommissure appears as a rounded band crossing the forepart of the third ventricle, and is traceable into the temporal lobes in two parts. The medicommissure, which consists mainly of cells, unites the thalami at the narrowest part of the diacœle at about the level of the aqueduct. The postcommissure is seen in the caudal portion of the diacœle just above the entrance to the aqueduct, to which it forms a curved roof. After passing through the thalami, its diverging fibres continue into the white substance of the hemispheres. Some are supposed to be connected with the pineal roots and with the nucleus of the third nerve, others with the quadrigeminal body.

FIG. 69.

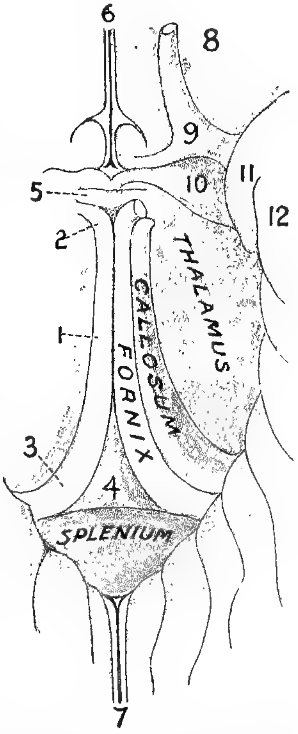


Scheme of the fornix: *A*, *A*, albicantia; *PPP*, precommissure; *F*, fimbria; *L*, lyre or psalterium; *V*, *V*, bundles of Vicq d'Azyr (so-called descending roots).

A commissure called the *supracommissure* unites the slight ridges known as the habena on the mesal surfaces of the thalami.

The Callosum.—The callosum is the great white commissural structure uniting the two cerebral hemispheres. It has been usually taught that its fibres associate identical areas of the two hemicerebrums; but late researches seem to show that this is not altogether true, although they certainly do this in part. In the process of brain development the callosum begins in front of the fifth ventricle, and later reaches backward as the hemispheres develop over the midbrain. The caudal or posterior extremity of the callosum is the *splenium;* its anterior end is the *genu,* and this after it has curved downward and backward is called the *rostrum* or beak. The body of the callosum is the part between the splenium and the rostrum. Its free portion forms the roof of the lateral ventricles. A horizontal transection of the cerebrum on a level with the callosum, as in Fig. 68, shows a well marked transverse striation; a striation is also present on its ventral surface. Obersteiner has given the name *indusium griseum,* rendered by Wilder simply as the mononym *indusium,* to this thin layer of gray substance above the callosum. This indusium, which is a continuation of the cortex of the callosal gyre (gyrus cinguli or gyrus fornicatus), was first recorded by Giacomini, although it was recognized in 1880 by Wilder, who failed to note and publish his observation. The researches indicating the transmission of nervous impulses by cell contact give to these as to other commissural striations a new significance.

Fig. 70.



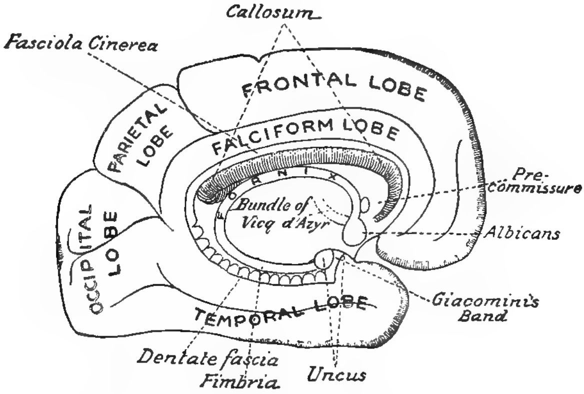
Ventral surface of the fornix and callosum and its intermediate surroundings; the brain trunk (or peduncle) has been removed from the base; the plane of section of the brain trunk is towards the left hemisphere: 1, body of fornix; 2, fornicolumn; 3, posterior pillar of the fornix; 4, exposed transverse fibres of the callosum; 5, precommissure; 6, anterior notch of the pallium or forebrain mantle; 7, posterior notch of the pallium; 8, olfactory tract; 9, trigonum (olfactory triangle); 10, precribrum (anterior perforated space); 11, basal view of the uncus; 12, cephalic part of the hippocampal gyre. (After Rauber.)

The Fornix.—The fornix is a body of complicated construction. Cephalad are its *fornicolumns* or *anterior pillars;* its two portions coming together in the middle line constitute its *body;* and these caudal portions, which soon separate from each other, passing to the right and the left, are its *posterior pillars.* The anterior pillars curve downward slightly, diverging in front of the porta, and pass-

ing through the tuber cinereum end in the albicantia at the base of the brain. The posterior pillars curve outward and downward over the thalami, and pass along the hippocamps, as narrow flat bands of white substance known as the *fimbriæ*. Sometimes the under or ventral surface of the fornix is designated as the *lyre* or *psalterium*, because of some fancied resemblance to harp strings; but this, as Wilder points out, is not a part, but merely a surface. Fig. 69 is a schematic representation of the fornix. The bundles of Vicq d'Azyr were formerly supposed to be direct continuations of the reflected anterior pillars, but this view is now regarded as doubtful.

The Dentate Gyre and Fasciola Cinerea.—The dentate gyre, or fascia dentata, is a notched or dentated gray cord at the inner side of the dentate or hippocampal fissure, in the bottom of which it is almost hidden—a low serrated ridge of gray in a narrow white valley. It follows the fissure or valley upward towards the sple-

Fig. 71.



Mesal surface of the left hemicerebrum of a fetus. (After Rauber.)

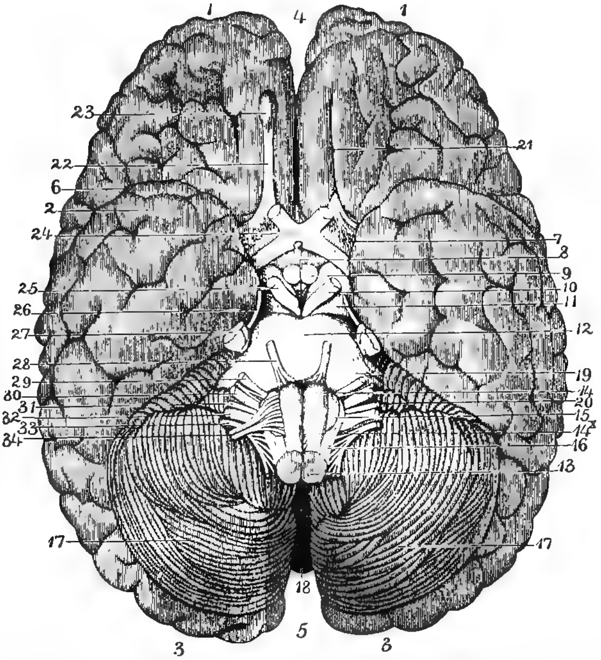
nium of the callosum, where it becomes continuous with the *fasciola cinerea*, a similar gray layer, but one having a smooth margin: these together represent a rudimentary gyre. These parts and various aspects of the commissures, callosum, fornix and related structures, notches, fissures, foramens, surfaces, and lobes, are well shown in Figs. 69, 70, and 71.

Base of the Brain.—In Fig. 72 are represented all the important structures seen at the base of the brain after the removal of the membranes, including portions of every great embryonal subdivision of the encephalon—forebrain, interbrain, midbrain, hindbrain, and afterbrain.

Albicantia.—The albicantia, known also as the corpora mammillaria, candicantia, and pisciformes, are two small, round, white bodies squeezed in between the crura behind the tuber cinereum. They are sometimes called the bulbs of the fornix, being formed by

its anterior pillars after descending to the base of the brain. While these bodies are in the main composed externally of white, internally they are made up of gray substance; and they are connected by a gray transverse commissure. Gudden has divided each albicans into a mesal ganglion of small cells and a lateral ganglion containing large cells. Edinger regards the albicantia as the place of separation between the forebrain and the interbrain. They have connections with the pons, and also with the anterior tubercle of the thalamus; and they also have a tegmental bundle, which can be traced back of the gemina to the aqueductal cinerea.

FIG. 72.



Base of the brain: 1, frontal lobes of the cerebrum; 2, temporal lobes; 3, occipital lobes; 4, 5, cephalic and caudal extremities of the great longitudinal fissure; 6, Sylvian fissure; 7, precribrum; 8, infundibulum; 9, albicantia; 10, postcribrum; 11, crura; 12, pons; 13, oblongata; 14, pyramid; 14x, decussation of the pyramids; 15, olive; 16, restiform body; 17, lateral lobes (hemispheres) of the cerebellum; 18, vermis at the bottom of the valley separating the latter; 19, medipeduncle; 20, floccule; 21, fissure which accommodates the olfactory lobe, 22; 23, bulb of the olfactory lobe; 24, chiasm; 25, oculomotor nerve; 26, trochlear nerve; 27, trifacial nerve; 28, abducent nerve; 29, facial nerve; 30, auditory nerve; 31, glossopharyngeal nerve; 32, vagus nerve; 33, accessory nerve; 34, hypoglossal nerve. (After Hirschfeld-Sappey.)

Tuber Cinereum.—The *tuber*, or tuber cinereum, in the interior of which is the cavity of the *infundibulum*, is a projection at the base of the skull, lying between the chiasm and the albicantia. Close examination shows on its outer side a tract of gray matter, which Meynert termed the basal optic ganglion, but this ganglion does not appear to be connected with the optic nerve, as was sup-

posed by Meynert. From it on each side a small tract issues which after crossing the middle line passes backward to the subthalamic body; by some this is supposed eventually to join the upper fillet, while in front it may be connected with the lenticula.

The Infundibulum.—The infundibulum appears as a conical hollow process, a continuation of the tuber cinereum, descending from the third ventricle behind the chiasm, to end in the posterior lobe of the hypophysis, in the process of brain development having grown downward from the floor of the forebrain or second cerebral vesicle.

FIG. 73.

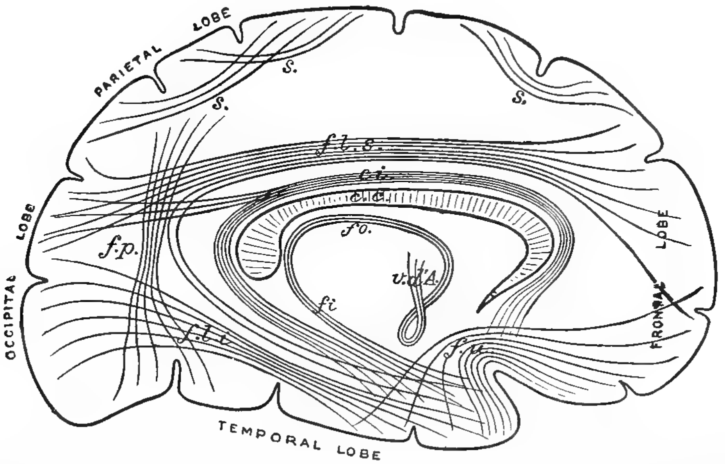


Diagram of association fibres of cerebrum: *s*, short fibres connecting adjacent gyres; *f.l.s.*, superior longitudinal, *f.l.i.*, inferior longitudinal, *f.u.*, uncinate (to the uncinate gyre), and *f.p.*, perpendicular fasciculus; *ci*, cingulum; *fo*, fornix; *fi*, fimbria; *v.d'A.*, bundle of Vicq d'Azyr. (After Schäfer-Meynert.)

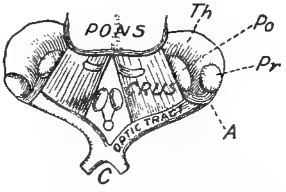
Association Fibres of the Cerebrum.—Various regions of the cortex of the hemispheres are associated functionally and of course anatomically: a general idea of the cerebral association fibres can be obtained from Fig. 73. Association fibres, called *arcuate*, which connect parts of the same hemisphere, are very numerous, and pass in many directions, uniting adjacent or near convolutions of the same lobe, or more distant parts of the same hemisphere. The subfrontal gyre, for example, is joined with portions of the temporal lobe; parts of the temporal and the occipital, of the frontal and the occipital, and of the parietal and the temporal, are connected. The fornix in part of its course connects a portion of the hippocampal convolution with the thalamus. *Commissural* association fibres which pass from one side of the brain to the other chiefly in the callosum or in the precommissure have already been considered. Some arcuate fibres associating areas of the same hemisphere pass in the cingulum, which lies within the cingulate convolution (callosal gyre or gyrus

fornicatus). The bundle of Vicq d'Azyr is a collection of nerve fibres which pass between the thalamus and the albicantia, while the fimbria or fringe has already been described as a prolongation of the posterior pillar of the fornix.

The Interbrain or Thalamencephalon.—The thalamencephalon, called also the interbrain or 'tweenbrain, includes the thalami, the genicula or geniculate bodies, the optic tracts, and the albicantia, although the entire thalamus is not always included. The thalamus is in many respects the most important ganglion of the nervous system, although its functions are still wrapped in considerable obscurity. Its surface is on the whole wedge shaped, with the blunt edge of the wedge in front. It presents several prominences, depressions, and bands or striæ, and is subdivided into at least three parts, the *anterior tubercle*, the *posterior tubercle* or *pulvinar*, and below and external to the pulvinar a *lateral* or *external tubercle* or *nucleus*. The anterior tubercle is compared by Edinger to a wedge driven in between the other two with its broad end in front. Other prominences, known as the *habenulas* or *pineal peduncles*, are connected on both sides with the pineal region. The *trigonum habenulæ* is a small triangular area in this part of the thalamus between the pulvinar, habenula, and midbrain. A thin layer of white matter covering the ventricular surface of the thalamus is called the *stratum zonale*, which consists of fine nerve fibres derived from the optic tract and retina, being in fact a thin expansion of the optic tract.

The Geniculate Bodies.—On the posterior inferior face of the pulvinar are seen two small rounded protuberances, called the *postgeniculum* and *pregeniculum*, or the *corpus geniculatum internum* and *corpus geniculatum externum*. The postgeniculum, or internal geniculate body, which is regarded by Obersteiner and others as part of the acoustic tract, is an oval eminence to which run fibres which have been traced as the hindermost layer of the chiasm into the corresponding bundle of the opposite optic tract. The pregeniculum, or external geniculate body, a slight eminence placed to the outer side of the pulvinar, also contributes fibres to the optic tract. These bodies are sometimes discussed as belonging to the thalamus, but may be regarded with the quadrigeminal body as parts of the mesencephalon or midbrain. They are connected by a loop or band of fibres. Their positions with reference to the pulvinar and to near structures are shown in Fig. 74.

FIG. 74.



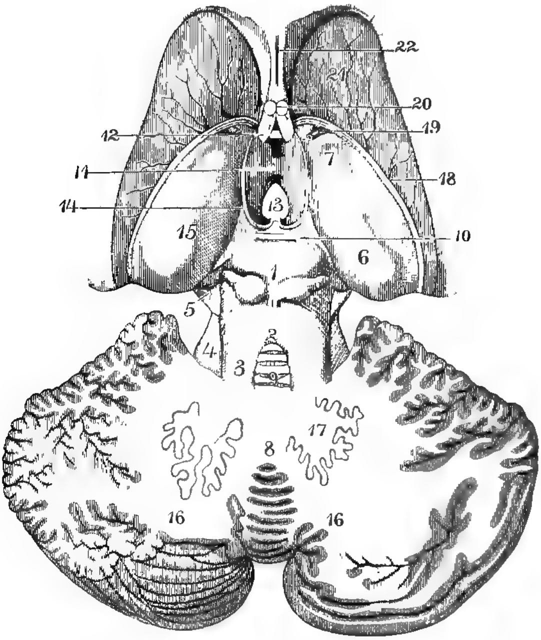
View of the base of the interbrain and midbrain: *Th*, thalamus; *Po*, postgeniculum (internal geniculate body); *Pr*, pregeniculum (external geniculate body); *A*, intergeniculate loop. (After Rauber.)

The Cavity of the Interbrain.—Between the thalami lies the third ventricle, or diacœle, the special cavity or chamber of the inter-

brain. The thalami are close together and trend inward somewhat towards the middle line, to some extent fusing, and thus forming the gray medicommissure. The aqueduct of Sylvius passes from this cavity to the metepicœle, or fourth ventricle; and its floor opens out, as already described, into a funnel-like depression which forms the entrance to the infundibulum.

The Conarium.—The conarium, or pineal body (Fig. 75), is included in the thalamencephalon, forming with its peduncles a portion of its roof. It has been shown that in the development of the roof of

Fig. 75.



Striata, thalami, quadrigeminal body, and cerebellum: 1, quadrigeminal body; 2, valvula (anterior velum); 3, prepeduncle; 4, upper portion of the medipeduncle; 5, upper portion of the crus; 6, pulvinar of the thalamus; 7, anterior tubercle; 8, vermis; 9, lingule; 10, postcommissure; 11, medicommissure; 12, precommissure; 13, conarium (pineal body) turned forward; 14, pineal peduncle; 15, thalamus; 16, hemispheres of the cerebellum; 17, dentatum (dentate nucleus); 18, tenia (semicircular band); 19, vein of the caudatum; 20, fornicolumn (anterior pillar of the fornix); 21, caudatum; 22, ventricular septum. (Hirschfeld and Sappey.)

the second cerebral vesicle a hollow median process is formed which becomes the conarium or epiphysis cerebri, which in some reptiles is a rudimentary eye occupying an opening in the middle of the skull. It is about the size of a cherrystone, usually of a reddish color, and is situated between the caudal portions of the thalami, its inferior surface resting in the mesal groove of the pregeminum, the splenium of the callosum having the under part of its curvature just above it and the cephalic end of the cerebellum abutting against it. While the conarium has little functional importance, its position is

such that a lesion of any size involving it might include three of the most important regions of the brain,—the interbrain, of which it is a part, and the cerebellum and the cerebrum, by which it is encompassed. The ancients attributed to the conarium, probably because of its central position, fanciful powers and virtues, even designating it as the seat of the soul. Its peduncles are flattened stalks of white fibres, each of which is separated by the pineal recess into a ventral and a dorsal portion, the former going to the postcommissure, and the latter, along the thalamus in the form of striæ, merging into the trigonum habenulæ. In the main the conarium is composed of epithelial tubules, but its peduncles contain a few nerve fibres; its follicles often contain gritty matter which is called *brain sand.*

The Hypophysis.—The hypophysis, pituitary body, or pituitary gland is a small reddish gray or reddish yellow mass, situated in the sella turcica of the sphenoid bone at the base of the brain. It was formerly described as a gland because it was supposed to secrete the pituita or mucus of the nose. It is composed of two distinct structures, a posterior or caudal lobe, in the walls of which are found nerve cells and fibres and neuroglia, and which is directly continuous with the infundibulum, and contains a cavity which was originally a part of the third ventricle. In lower animals at least it may be regarded as a true subdivision of the brain. In higher vertebrates and in man the nervous structures become almost obliterated. The anterior or cephalic lobe is not a nervous structure, but has developed as an epithelial prolongation of the cavity of the mouth. In adult man it contains epithelial tubes, connective tissue, and bloodvessels from the pia, and here and there the tubules are cut in two so as to form vesicles. Microscopically this portion of the hypophysis resembles the thyroid body; and in its alveoli or tubules are found a colloid material and also a special pigment.

Subthalamic Region.—A transection of the brain through the pulvinar at the postcommissure, as shown diagrammatically in Fig. 76, will bring into view several gray or dark masses and various related tracts. The structures of this subthalamic region have been studied by Luys, Forel, Flechsig, Wernicke, and Edinger, among others, who have determined many facts with reference to their anatomy, their histology, and their relations to other structures, although their functions still remain practically unknown. The two chief structures are the *red nucleus* and *subthalamus;* and a little posterior to these a transection reveals the *intercalatum* or *substantia nigra,* shown in the diagram as if in the same plane. Other parts shown are the postcommissure, the iter, or aqueduct, the anterior nucleus of the third nerve, the caudatum, lenticula, and internal capsule, the pes pedunculi, the optic tract, and the *ansa* or lenticular bundle (ansa lenticularis or lenticular loop). Various names have been applied to the masses of fibres or to portions of them in this region, as the

Fig. 76.

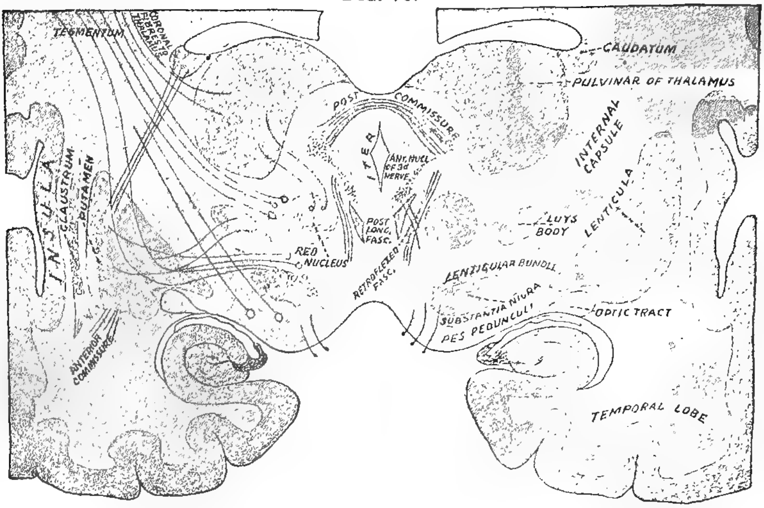
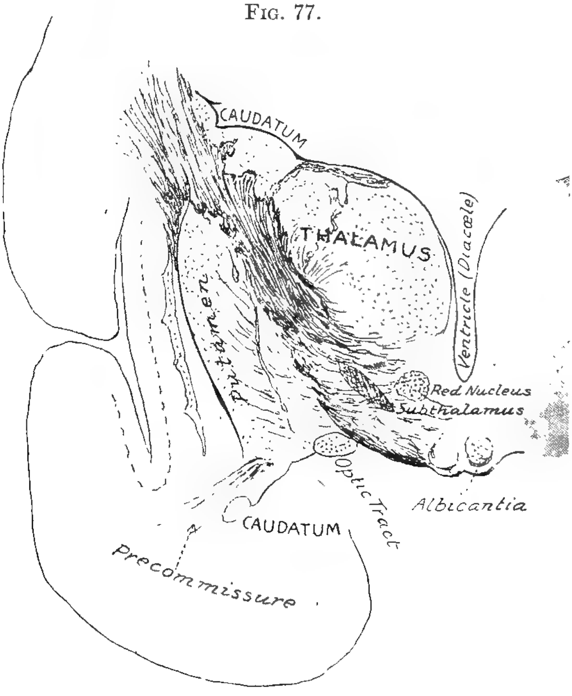


Diagram of a section through the vicinity of the postcommissure, showing the ganglia and the course of some of the fibres of the subthalamic region. (After Edinger.)

stratum intermedium, the *substantia innominata*, the *zona incerta*, terms which simply indicate the uncertainty and paucity of our knowledge.

Fig. 77.



Transection showing the subthalamic region from a child four weeks old. (Edinger.)

In Fig. 77 are shown some details of a section from a child four weeks old, which gives a more correct idea of the natural appearances

than is obtained from the diagram, which is not quite accurate in details and arrangements. The red nucleus and subthalamic body are exposed by this section.

Red Nucleus.—The red nucleus, nucleus ruber, or tegmental nucleus, is an ovoid, brownish gray mass, extending backward and just below the thalamus into the midbrain below the aqueduct. It is round in transections and of an elongated oval shape in sagittal sections. Fibres of the coronal radiation surround about one third of this nucleus, which contains numerous multipolar angular cells.

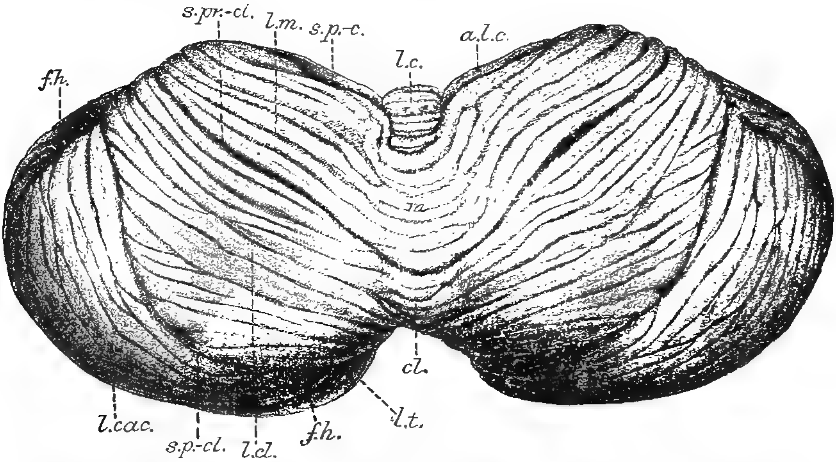
Subthalamus.—The subthalamus, subthalamic ganglion, or body of Luys, is a small mass of yellowish gray color in the fresh brain, situated just below the thalamus. On transection it appears as a spindle with the greater convexity placed dorsally. It lies just between the tegmentum and the crusta. It is composed of a network of fine medullated fibres throughout which multipolar nerve cells of moderate size are distributed.

The Quadrigeminal Body.—The quadrigeminal body (quadrigeminum), the chief constituent of the midbrain, is conspicuous early in embryonic life. Considered as a whole it is quadrate in shape, and is subdivided on its dorsal surface into four rounded white masses. It is situated beneath the splenium of the callosum in the interval between the cerebrum and the cerebellum. Its cavity is the mesocœle, or aqueduct of Sylvius, the four elevations of which it is composed constituting the main roof of the mesocœle. The quadrigeminal body has most important connections with the optic, auditory, and other cranial nerves, with the cerebellum, and with other parts.

The Cerebellum.—The cerebellum consists of two lateral lobes, usually called hemispheres, joined by a narrow lobe, the *worm* or *vermis*, which is subdivided into an upper, superior, or cephalic portion, the *prevermis*, and a lower, inferior, or caudal portion, the *postvermis* (Figs. 78, 79, and 80). In several figures already given, profile and other partial views of the cerebellum have been shown. It is situated dorsally to the fourth ventricle and both behind and below the quadrigeminal body. By its three sets of peduncles it is connected directly with the quadrigeminal body, with the oblongata, and with the pons. Between its peduncles, dorsally to the cephalic portion of the fourth ventricle, a white lamina, the *valvula* (*valve of Vieussens*, *anterior* or *superior medullary velum*), extends from the vermis to the postgeminum, or testes; while the more caudal portion of the ventricle is roofed by another smaller semilunar lamina, the *posterior* or *inferior medullary velum*, or *valve of Tarini*—sometimes called the *commissure of the flocculus*—which is continued downward to the upper extremities of the posterior columns of the spinal cord. The fissures of the surface of the cerebellum are closer, more regular, and more parallel than those of the cerebrum, giving a foliated appearance. The largest fissure (*f.h.*, in Figs. 78 and 79), known as

the *great horizontal fissure* or *peduncular sulcus* (Wilder), divides the surface into an upper and a lower portion. The cerebellar lobes or hemispheres are separated by anterior and posterior notches. Sections across the direction of the lamina reveal an arrangement of gray and white matter which has a tree-like appearance and is known as the *arbor* or *arbor vitæ*. This appearance is due to the subdivision of the lamina into smaller and these into still smaller branchings. The central white matter of the vermis, regarded as a whole, is sometimes called the *corpus trapezoides*, or *trapezoid body*, which is also applied to an important structure in the oblongata preferably designated the *trapezium*.

Fig. 78.



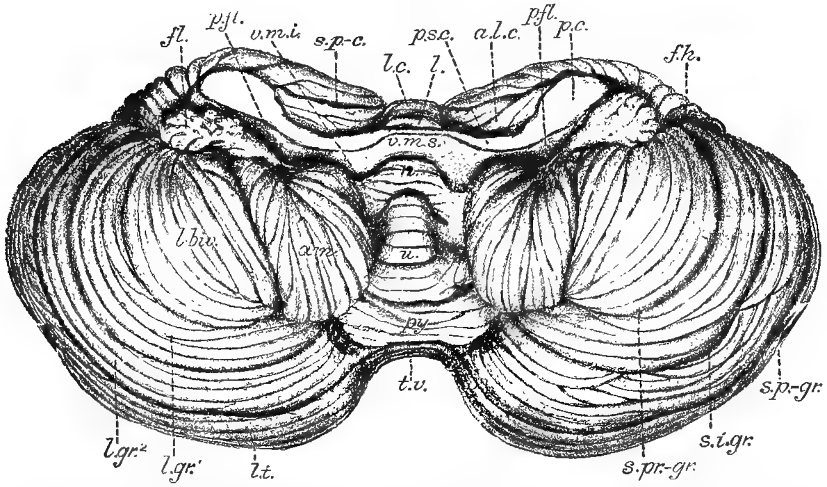
View of the upper surface of the cerebellum, natural size, from a photograph: *l.c.*, lobulus centralis (central lobule); *a.l.c.*, ala lobuli centralis (ala or lateral portion of central lobe); *m.*, culmen monticuli (monticulus); *l.m.*, lobus culminis (culmen); *cl.*, clivis (declivity): *l.cl.*, lobus clivi (quadrangular lobe); *l.cac.*, lobus cacuminis (presemilunar lobe); *l.t.*, lobus tuberis (postsemilunar lobe); *s.p.-c.*, sulcus postcentralis (postcentral fissure); *s.pr.-cl.*, sulcus preclivalis (preclival fissure); *s.p.-cl.*, sulcus postclivalis (postclival fissure); *f.h.*, *f.h.*, fissura horizontalis magna (great horizontal fissure, peduncular sulcus). (Schäfer, in Quain's Anatomy.)

Subdivisions of the Cerebellar Surface.[1]—The upper surface of the cerebellum is less complex than the lower, the vermis merging into the lateral lobes or hemispheres. The prevermis, or superior vermiform process, is usually divided from the anterior to the posterior notch into a succession of lobes, namely, the *central lobe*, and the *monticulus*, subdivided into two parts, an anterior elevation, called the *culmen*, and a posterior slope, the *declive* (*declivis* or *clivis*). A subdivision called the *cacumen* is sometimes present below the declive, and the part connecting the hemispheres in the posterior

[1] Cerebellar terminology illustrates forcibly the confusion caused by a multiplication of synonyms for the same part, and it is to be hoped that soon a uniform system of names—mononyms whenever possible—will be generally adopted.

notch is spoken of as the *commissura simplex* or *simple commissure*. The lobes or lobules of the hemisphere on this aspect correspond closely to the subdivisions of the vermis; and the names given in the illustration have reference to these relations, as *lobus culminis*, *lobus clivi*, *lobus cacuminis*, and *lobus tuberis*. Fissures of similar names bound and separate these lobes. While on the upper surface of the cerebellum but little demarcation exists between the vermis and the lateral lobes, on the lower these lobes or hemispheres are separated by a broad depression, the *vallecula* or *valley*, which extends from before backward along the postvermis. The subdivisions of the

FIG. 79.

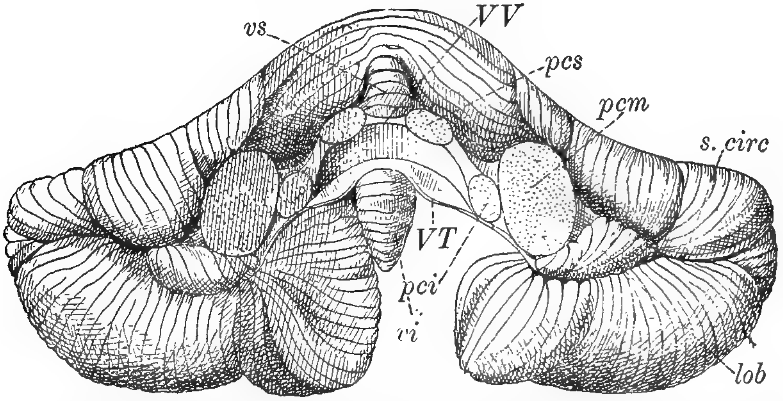


View of the lower surface of the cerebellum, natural size, from a photograph: *l.*, lingula; *l.c.*, lobulus centralis (central lobule); *a.l.c.*, ala lobuli centralis (ala or lateral portion of the cerebral lobe); *s.p.-c.*, sulcus postcentralis (postcentral fissure); *v.m.s.*, velum medullare superius (valvula); *p.s.c.*, pedunculus cerebelli superior (prepeduncle); *p.c.*, pedunculi cerebelli medius et inferior (medipeduncle and postpeduncle); *n.*, nodulus; *v.m.i.*, velum medullare inferius (valve of Tarini); *p.fl.*, pedunculus flocculi (peduncle of the flocculus); *fl.*, flocculus; *u.*, uvula; *am.*, amygdala (tonsilla); *py.*, pyramis; *l.biv.*, lobus biventralis (cuneiform or digastric lobe); *t.v.*, tuber valvulæ seu posticum (tuber); *l.t.*, lobus postero-inferior (inferior semilunar or postsemilunar lobe); *l.gr.*[1], lobus gracilis anterior (anterior slender lobe); *l.gr.*[2], lobus gracilis posterior (posterior slender lobe); *s.pr.-gr.*, sulcus pregracilis (pregracile fissure); *s.i.-gr.*, sulcus intragracilis (intragracile fissure); *s.p.-gr.*, sulcus postgracilis (postgracile fissure); *f.h.*, fissura horizontalis magna (great horizontal fissure, peduncular sulcus). The vallecula has been somewhat opened, to display the parts of the lower worm. (Schäfer, in Quain's Anatomy.)

postvermis or inferior worm are the *central lobe*, *lingula*, *nodule*, *uvula*, *pyramid*, and *tuber* (*tuber valvulæ seu posticum*); those of the lateral lobes or hemispheres are the *flocculus* (*pneumogastric lobe*), *tonsil* (*amygdala*), *biventral lobe* (*digastric* or *cuneiform lobe*), *slender lobe* (*lobus gracilis*),—which last may be subdivided into the *lobulus gracilis anterior* and *lobulus gracilis posterior*, with their fissures of similar name,—and finally the *postero-inferior lobe* (*inferior semilunar* or *postsemilunar lobe*). The continuity of the vermis with the hemispheres is not as obvious upon the lower as upon the upper aspect of the

cerebellum ; still the mesal and lateral parts can be related to each other with but little difficulty,—the nodule to the flocculi, the uvula to the tonsils, the pyramid to the biventral lobes, the tuber to both the slender and the postero-inferior lobe ; while the lateral or hemispheric portions of the lingula and central lobe are rudimentary. Besides the lobar divisions just given, the upper surface of the hemispheres is sometimes divided into an anterior square, or *quadrangular lobe,* which extends from the anterior notch backward as far as the posterior limits of the vermis, and a posterior or *presemilunar lobe* (*superior semilunar lobe*), which lies between the quadrangular lobe and the great horizontal fissure. The quadrangular and semilunar lobes appear in the views on both surfaces of the cerebellum, but the latter on its lower or caudal aspect is called by Wilder the *postsemilunar lobe.* In studying the cerebellum confusion may be caused

FIG. 80.



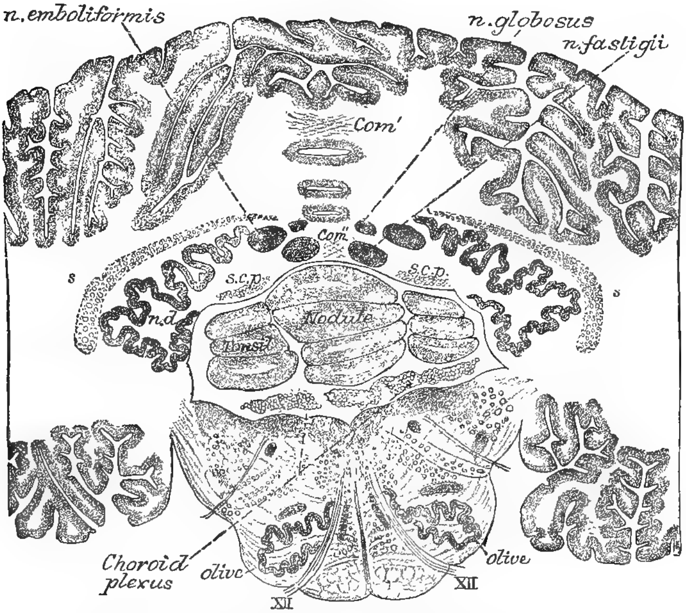
Cerebellum seen on its anterior surface : *vs,* anterior extremity of the prevermis (superior vermis) ; *VV,* valvula ; *pcs,* section of the prepeduncle ; *vi,* anterior extremity of the postvermis (inferior vermis) ; *pcm,* section of the medipeduncle ; *pci,* section of the postpeduncle ; *VT,* posterior medullary velum (valve of Tarini) ; *lob,* flocculus (pneumogastric lobule or lobe) ; *s. circ,* circumferential border. (Van Gehuchten.)

through consulting different books, not only because of terminology, but also because of the different planes in which the views are presented. In some of the illustrations the tuber does not appear as a part of the upper surface. In Fig. 79 it is represented below the pyramid, in the view of the lower surface, while in the figures of Edinger and Wilder it appears on the dorsal or upper surface. In Fig. 80 is a view by Van Gehuchten of the anterior surface of the cerebellum which differs from those usually given, presenting in the mesal line, above and below, the anterior extremity of the prevermis *vs,* resting on a thin white layer. The valvula is seen extending transversely between the prepeduncles, and under this structure is a sort of cul de sac of the fourth ventricle.

Ganglia and Deposits of the Cerebellum.—The most impor-

tant nuclei of the cerebellum (shown in Fig. 81, with some structures of the oblongata) are the *dentatum* (*nucleus dentatus*, *corpus ciliare*, *cerebellar olive*), the *embolus* (*nucleus emboliformis*), the *globulus* (*nucleus globosus*), and the *fastigatum* (*nucleus fastigii*, *nucleus of the roof*, or *tegmental nucleus*). The position of the dentate nucleus, the largest ganglion of the cerebellum, is shown at *nd.* The embolus lies close to the mesal side of the dentatum, and probably belongs to it, while the globulus is a small distinct deposit internal to the embolus. The fastigatum is an important collection of cells which lies close to the median line in the white matter of the postvermis.

FIG. 81.



Section across the cerebellum and oblongata, showing the position of the nuclei in the medullary centre of the cerebellum: *n.d.*, dentatum (nucleus dentatus); *s*, band of fibres derived from the restiform body, partly covering the dentatum; *s.c.p.*, commencement of prepeduncle (superior cerebellar peduncle); *com'*, *com''*, commissural fibres crossing in the median white matter. (Quain's Anatomy, after Stilling.)

The epicœlian portion of the fourth ventricle is shaped somewhat like a tent or gable roof, as can be seen in several profile views already given, and the fastigatum is in the wall of the roof of this extension; hence one of its names, *roof nucleus*. These cerebellar deposits are here and there united imperfectly with one another. The dentate and emboliform nuclei have much the same microscopic structure, while the roof and globose nuclei are similar in structure, having larger cells than the two former.

Cerebellar Conducting Tracts.—Persevering efforts have been made to trace the course of the fibres which pass to and from and

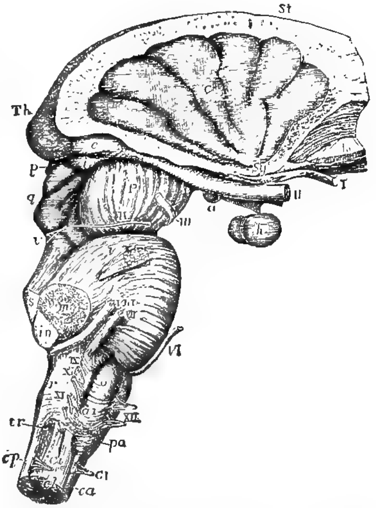
through the cerebellum. Stilling, and more recently Edinger, Marchi, Bruce, and others, have devoted much labor to the study of this and other problems connected with the cerebellum. Several sets of fibres which have been well traced are the *intraciliary*, which pass from the anterior peduncles into the dentatum, or ciliary body; the *extraciliary*, which pass external to the dentatum and converge towards it, partly entering and partly passing through its gray matter; and the *dendritic tracts*, sets of fibres which, arising from all parts of the cortex and diverging from each other like the limbs of a tree, pass into the white substance. The mass of fibres which constitutes the *extraciliary* tracts is sometimes called the *fleece*, because of its resemblance to wool. The restiform bodies are in large part connected with the fleece. A band or tract called the *direct sensory tract* of the cerebellum arises near the globose nucleus and passes to the outer wall of the fourth ventricle, some of its fibres passing out with fibres of sensory cranial nerves, and others going to the posterior columns of the oblongata and spinal cord. In the cephalic portion of the alba of the vermis is a most important encephalic decussation, the *anterior decussating commissure* of the cerebellum; a *posterior commissure* also passes through the posterior extremity of the vermis, connecting the hemispheres; and a *longitudinal commissure* is present in the vermis.

Fibres of the Cerebellar Peduncles.—With reference to the fibres of the cerebellar peduncles and their relation to the rest of the nervous system, Marchi concludes that the prepeduncles do not cross completely, but give some fibres to the thalamus of the same side; most of their fibres go to Stilling's nucleus of the opposite side; and no fibres go either to the optic tract or to the bundle of Reil. The medipeduncles are not solely commissural, but contain fibres which join the gray matter on both sides. The fibres in the postpeduncle for the opposite olive are both afferent and efferent. The dorsal longitudinal bundle and the fillet receive fibres from the cerebellum, chiefly in the vermis, and connect the nuclei of the cranial nerves with the cerebellum. The fillet connects the cerebellum with the pons, the quadrigeminum, and perhaps the striatum. The dorsal longitudinal bundle joins the fillet about the level of the olive, and both go into the anterolateral tracts, and probably are thus connected directly with the ventral horn and indirectly with the ventral roots. The origin of all these peduncles is spread over the whole gray matter, but the prepeduncles are connected chiefly with the dentate nucleus, the medipeduncles with the vermis. Schemes by Flechsig, Van Gehuchten, and Bruce, showing the most important tracts of the cerebellum, will be given later.

Crura, Pons, and Oblongata.—The crura, the great highways to the cerebrum, with the iter and quadrigeminal body, are constituents of the midbrain or mesencephalon, as has been already shown, but, for practical reasons, in the study of local diagnosis

and of various encephalic associations it is sometimes desirable to consider the crura, pons, and oblongata together. In Fig. 82 is an excellent view of these important structures and central parts of the cerebrum, as seen from the right side; and in other illustrations already given various aspects of these structures have been shown. The description accompanying the figure is sufficient to make clear all that is necessary regarding superficial appearances. The pons and oblongata, which serve to join together the forebrain, interbrain, hindbrain, and spinal cord, were often termed by older writers the *isthmus*. *Medulla oblongata* in the days of Willis included not only what is now known as the oblongata, but also the pons and the crura. The pons is made to include the entire thickness of the mass between the crura and a section through the well defined cephalic extremity of the ventral face of the oblongata; but it is more correct to regard all the dorsal portion of this segment as belonging to the oblongata—with Wilder designating it the *preoblongata*—calling all the more caudal portion of the isthmus the *postoblongata*.

FIG. 82.



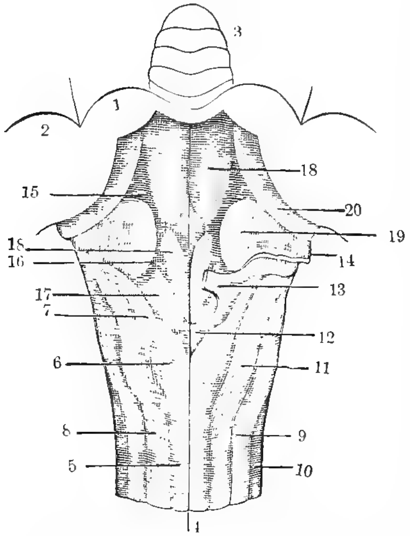
View of the oblongata, pons, crura, and central parts of the encephalon from the right side: *St*, upper surface of the striatum; *Th*, pulvinar of the thalamus; *C*, insula; *Sy*, fissure of Sylvius, in which are seen the white striæ of the olfactory tract; I, the olfactory tract; II, optic nerves a little in front of the chiasm; *a*, albicans with the tuber cinereum and infundibulum in front of it; *h*, hypophysis; *e*, pregeniculum, and *i*, postgeniculum; *P*, peduncle or crus of the cerebrum; III, right oculomotor nerve; *p*, epiphysis; *q*, quadrigeminum (quadrigeminal body); IV, trochlear nerve arising from *v*, the valve of Vieussens; V, placed on the pons above the right trigeminal nerve; *s*, the superior, *m*, the middle, and *in*, the inferior peduncle of the cerebellum cut short; VI, the sixth nerve; VII, facial nerve; VIII, auditory nerve; IX, glossopharyngeal nerve; X, pneumogastric nerve; XI, the uppermost fibres of the spinal accessory; XII, hypoglossal nerve; *pa*, pyramid; *o*, olive; *ar*, arciform fibres; *r*, restiform body; *tr*, tubercle of Rolando; *ca*, ventral or anterior, *cp*, dorsal or posterior, and *cl*, lateral columns of the spinal cord; *CI*, *Ci*, ventral and dorsal roots of the first cervical nerve. (Quain's Anatomy, after Allen Thompson.)

External Features of the Ventral Aspect of the Oblongata.—The length of the ventral surface of the oblongata (the postoblongata) is nearly an inch; its greatest breadth is about three fourths of an inch, and its thickness is a little less. On this surface the chief superficial structures are the *pyramids* or *anterior pyramids*, the *lateral columns*, and the *olives*. The anterior pyramids, prominences on each side of the mesal fissure, are largely composed of fibres which cross below at the decussation of the pyramids. The lateral columns are continuations of the anterior and to a small extent of the lateral columns of the cord, the main bulk of the fibres

of the latter crossing and becoming continuous with the anterior pyramids; so that, in the main, anterior and lateral and lateral and anterior change places in the spinal cord and oblongata. The anterior pyramids are frequently crossed by arched fibres, constituting the *ponticulus* (little bridge) of Arnold. The olives, known also as the *lower olives* or *olivary bodies*, appear externally as longitudinal oval eminences projecting from the lateral columns close to the pons and extending longitudinally about half the length of the oblongata. In the longitudinal grooves between the different structures of the ventral and lateral surfaces of the oblongata, and in the transverse furrow between the latter and the pons, the cranial nerves from the sixth to the twelfth have their origins, most of them by numerous roots.

External Features of the Dorsal Aspect of the Oblongata.—On its dorsal aspect the oblongata includes both the postoblongata and the preoblongata—the latter lying dorsally to the pons—the length of the entire structure being about two inches, and the depth of the preoblongata being about half that of the postoblongata at the latter's greatest thickness. This aspect of the oblongata presents the *floor of the fourth ventricle* or *metepicœlian floor*, and two important prolongations of the columns of the spinal cord, the *postpyramids* and *restes* (*posterior pyramids* and *restiform bodies*). When the columns of Goll or posteromedian tracts of the spinal cord reach the oblongata, they become demarcated by the median fissure and a slight lateral groove, as narrow cylinders, the *gracile funicles* (*funiculi graciles*), which cephalad expand into elevations known as the *clava*, the two together constituting the postpyramids of the postoblongata. Between the funiculi graciles and posterolateral grooves are other distinct columns, known as the *cuneate funicles* (*funiculi cuneati*), which are prolongations of the posterolateral columns of Burdach; and still more laterally are the *funiculi of Rolando* or *external cuneate funicles*, with

Fig. 83.

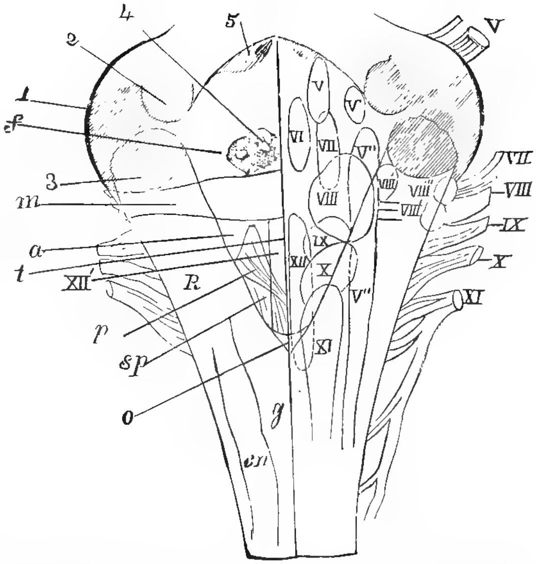


Dorsal part of oblongata and floor of fourth ventricle; magnified one and a half times: 1, prepeduncle of the cerebellum, cut surface; 2, medipeduncle and postpeduncle; 3, lingula lying on the valvula or velum; 4, anterior or ventral median fissure; 5, gracile funicle; 6, clava tapering away at 7; 8, internal cuneate funicle; 9, external cuneate funicle; 10, lateral funicle; 11, cuneate eminence; 12, obex; 13, ponticulus; 14, inferior velum; 15, prefovea (superior fovea); 16, postfovea (inferior fovea); 17, ala (ala cinerea); 18, terete funicle; 19, auditory eminence crossed by auditory striæ; 20, root of auditory nerve. (Leidy.)

their *tubercles*, which are prolongations and projections of the substantia gelatinosa of the cord. Another narrow dorsolateral strand, the *direct cerebellar tract*, appears on each side just above the tubercle of Rolando. Arched fibres course over the pyramids, olives, and cuneate funicles from the ventral median fissure. The restiform body is formed by the coming together of these columns and bands —the cuneate funicles, funicle of Rolando, direct cerebellar tract, and external arched fibres—with the addition of other fibres coming from the opposite side of the oblongata; and these restiform bodies become the postpeduncles or inferior peduncles of the cerebellum.

The Floor of the Fourth Ventricle.—The dorsal aspect of the oblongatas is largely constituted by the *floor of the fourth ventricle*, which is rhomboidal, lozenge shaped, or diamond shaped, as shown in several views. The fourth ventricle as a cavity has already been described; but it is of practical advantage to be familiar with the appearances presented by the floor of this space, and to know the meaning of its eminences, depressions, grooves, striations, and colorations. In the first place, this metepicœlian floor presents a shallow *mesal groove* or *sulcus;* and *white acoustic* or *medullary striæ*, which divide it into a cephalic or upper and a caudal or lower portion. By a comparison of Figs. 83 and 84 with the description in the text and with the legends, the landmarks can be learned. Because it has the shape of a pen, the most caudal portion of the ventricle has been called the *calamus*

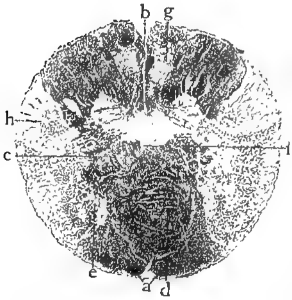
FIG. 84.



View of the dorsal surface of the oblongata. The left half of the figure represents: *Cn*, funiculus cuneatus, and *g*, funiculus gracilis; *O*, obex; *sp*, nucleus of the spinal accessory; *p*, nucleus of the pneumogastric; *p + sp*, ala (ala cinerea); *R*, restis (restiform body); XII′, nucleus of the hypoglossal; *t*, funiculus teres; *a*, nucleus of the acusticus; *m*, striæ medullares; 1, 2, and 3, cerebellar peduncles; *f*, prefovea; 4, eminentia teres; 5, ceruleum (locus ceruleus). The right half of the figure represents the nerve nuclei diagrammatically: V, motor trigeminal nucleus; V′, median and V″, inferior sensory trigeminal nuclei; VI, nucleus of abducens; VII, facial nucleus; VIII, posterior median acoustic nucleus; VIII′, anterior median, VIII″, posterior lateral, VIII‴, anterior lateral acoustic nuclei; IX, glossopharyngeal nucleus; X, XI, and XII, nuclei of vagus, spinal accessory, and hypoglossal nerves respectively. The Roman numerals at the side of the figure, from V to XII, represent corresponding nerve roots. (After Erb.)

scriptorius; while its pointed lateral extremities, at the position of the medipeduncles, are the *lateral recesses.* In the upper segment or triangle of the floor, near the aqueduct, on both sides of the median line is a slate-blue area, the *locus ceruleus,* corresponding to the subjacent *substantia ferruginea,* which contains pigmented cells from which it is supposed that the trophic fibres of the trigeminal nerve are derived. On each side of the mesal sulcus run slightly prominent columns, the *funiculi teres* or *terete funicles,* fasciculi or bundles which are dorsad to the dorsal or *posterior longitudinal* bundles of the tegmens. In the lower portion of the superior triangle these rounded columns become slight elevations, the *eminentia teres,* which are in relation with the nuclei of the abducens nerve and the curved rootlets from the facial cell nest which pass over the abducens nuclei and form what is known as the *genufacialis.* External to the terete eminence is a small angular depression, the *prefovea* or *superior fovea.* Below the medullary striæ, in the lower triangle of the ventricular floor, a little laterad of the mesal groove, is another small triangular depression, the *postfovea* or *inferior fovea.* A triangular area of a gray or ash color in the lower portion of the caudal subdivision of the ventricular floor is known as the *ala* or *ala cinerea.* This area is the darkest portion of the floor; it corresponds in part to the nuclei of the pneumogastric and of the glossopharyngeal nerve. At each lateral extremity of the medullary striæ is a thin grayish ridge, the *teniola cinerea,* which has been shown by Spitzka and others to correspond to the *tuberculum acusticum* in lower mammals. Between the medullary striæ, the mesal groove, and the ala cinerea is a white field, the *ala alba medialis,* which is related to the hypoglossal nucleus. It will thus be seen how these and other landmarks serve to fix the positions of structures in the interior of the oblongata, and therefore constitute important aids and guides in making either gross or microscopical examinations of these parts.

Fig. 85.



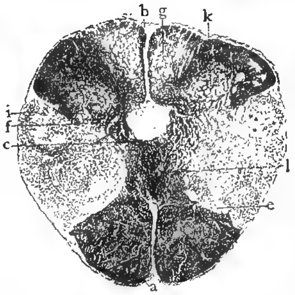
Section of the lower end of oblongata at level of decussation of anterior pyramids: a, anterior pyramidal tracts; b, posterior median septum; c, fibres of crossed pyramidal tracts crossing (d) to anterior pyramid of opposite side; e, ventral horn of gray matter isolated by decussating fibres; f, remains of bases of ventral horns; g, nucleus gracilis; h, enlarged and displaced posterior horns.

The Region of Transition from the Spinal Cord to the Oblongata.—The gray matter of the spinal cord retains the same general shape and appearance from the conus until the oblongata is nearly reached; but the gray substance of the posterior horns, as seen in sections, increases considerably as it is followed upward, and at about the level of the first, and sometimes even of the second

cervical nerve, these horns become club-shaped gray masses united with the central cinerea by only a thin stalk. A little higher, projections or tubercles are formed and bundles of white fibres begin to penetrate and pass through these gray masses. Two separate nuclei are here formed, known respectively as the *nucleus gracilis* and the *nucleus cuneatus*, which are in fact simply extensions upward of the spinal cinerea. These masses cephalad form the distinct elevations or eminences known as the *clavate* or *gracile tubercle* and the *cuneate tubercle* or *eminence*. The clavate nucleus is nearer than the cuneate to the posterior median groove. Remains of the posterior horns are still seen, and are known at this position as the *substantia gelatinosa of Rolando*. External to the cuneate nucleus is a small gray mass called the *external* or *accessory cuneate nucleus*, which probably represents a continuation of Clarke's column.

Interior Structures of the Oblongata.—In Figs. 85 and 86, the interior structures of most of the parts in the transitional region above mentioned are shown, and also two great decussations—one motor, composed of fibres of the anterior pyramidal tracts, and the other sensory, of fibres emerging from the cuneate and clavate nuclei. The oblongata is a complex structure, on the unravelling of which much labor has been spent, and not unfruitfully, although much still remains to be done. In the first place, all the nuclei or cell nests of the cranial nerves from the third to the last are located in the preoblongata and postoblongata, near their dorsal surfaces. The oblongatas also contain certain special ganglia, numerous intranuclear and intraganglionic tracts, and other long nerve tracts, which connect the spinal cord with the midbrain, the interbrain, the cerebrum, and the cerebellum. In all essential features the oblong cinerea corresponds with the gray matter of the spinal cord. Cell masses and eminences take the place of horns and elongated prominences of isolated columns. Motor nidi contain multipolar cells like those found in the ventral horns of the cord, and the nuclei of the sensory cranial nerves have a structure similar to that of the dorsal ganglia and dorsal spinal horns. It is only by studying many transverse, longitudinal, and horizontal sections that a familiarity with the internal structure of the oblongata and pons can be obtained. Fig. 87 is an illustration of the interior appearances seen in a tran-

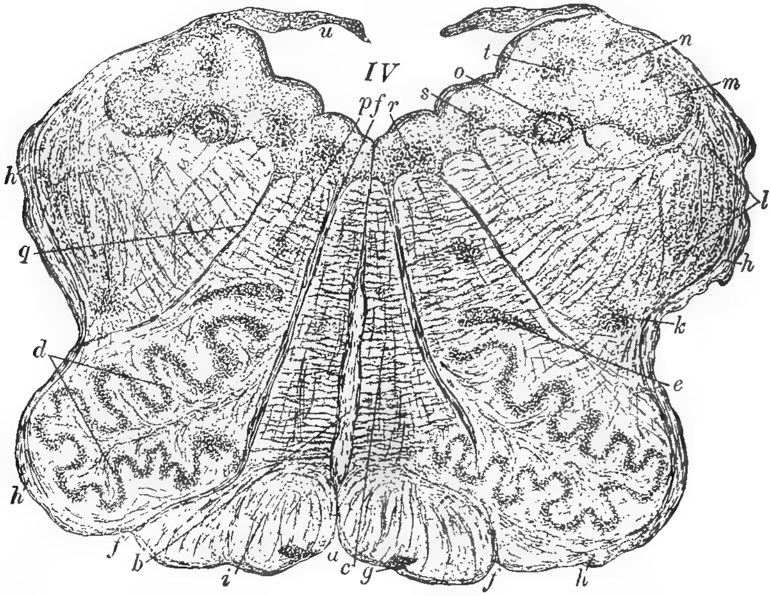
Fig. 86.



Section of oblongata at level of sensory decussation: a, anterior pyramidal tracts; b, posterior median septum; c, gray matter representing bases of ventral or anterior and dorsal or posterior cornua; e, pyramidal nucleus; f, sensory fibres displacing posterior horn; g, nucleus gracilis; i, posterior horn, substantia gelatinosa of Rolando; k, nucleus cuneatus; l, decussating sensory fibres. The decussation of the motor fibres is here complete.

section of the oblongata through the olives. Within each olive is an olivary nucleus, a lamina of gray matter of ovoid shape and of corrugated outline, having the appearance of a pouch with its mouth directed inward. In this zigzag zone of gray matter which forms the outer boundary of the open pouch are found numerous small multipolar cells mixed with neuroglia. Besides the olives the most important structure is the formatio reticularis; the other nuclei, fibres, grooves, and bundles have been or will be explained in other connections.

FIG. 87.

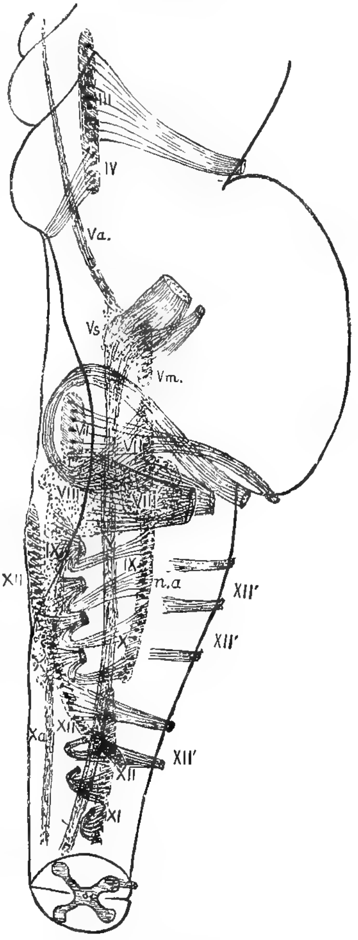


Section of oblongata of child through olivary bodies: *a*, anterior median groove; *b*, raphe; *c*, formatio reticularis; *d*, gray matter of nucleus dentatus of olive; *e*, dorsal accessory olivary body; *f*, root fibres of hypoglossal nerve; *g*, nucleus arciformis; *h*, external arcuate fibres; *i*, anterior pyramidal tract; *k*, remains of nucleus lateralis; *l*, substantia gelatinosa of Rolando and fibres of descending trifacial root; *m*, *n*, gray matter of posterior (cuneate) funiculus; *o*, funiculus solitarius; *p*, nucleus ambiguus; *q*, root fibres of pneumogastric nerve; *r*, *s*, hypoglossal and vagus nuclei; *t*, nerve cells of funiculus gracilis; *u*, posterior medullary velum closing in fourth ventricle, *IV*. (Piersol.)

Formatio Reticularis.—The *formatio reticularis*, or *reticular field*, consists of numerous interlacing fibres of different lengths interspersed with multipolar cells, and can be readily followed as a structure of peculiar appearance in the central regions of the oblongata and pons from the top of the posterior columns cephalad all the way into the midbrain. The reticular field is shown in several transections, and its great longitudinal extent in Flechsig's diagram of the encephalospinal conducting paths. In the oblongata it lies just dorsad of the pyramids and olives, and in the pons ventrad of most of the nerve nuclei, although some of these, as the nucleus of the facial and of a portion of the trigeminal, lie within its bounds; it also contains

a considerable collection of cells, called the *superior olive.* Just at the dorsal border of this formation, close to the raphe, an important angular tract known as the *dorsal* or *posterior longitudinal bundle* is isolated, and extends the whole distance from the spinal cord to the quadrigeminal body.

FIG. 88.



Profile view of the right half of the oblongata and pons, showing the chief cranial nerve nuclei and the course of their root fibres. The numbers indicate nerves of corresponding number: Va, mesencephalic root of the fifth nerve, probably passing to the motor root; V, descending (spinal) root of the fifth nerve (formerly called the ascending); Vs, its sensory, and Vm, its motor nucleus; VIII' is the outer or ventral nucleus of the eighth nerve; n.a is the nucleus ambiguus. (Quain's Anatomy.)

Interior Structure of the Pons.—The pons, as it has been delimited in this chapter, contains numerous small aggregations of gray matter, the *nuclei pontis;* but it is composed chiefly of masses of fibres, longitudinal and transverse. The longitudinal are mainly continuations of the pyramidal tract from the cerebrum to the spinal cord; but at least three sets of transverse fibres are easily recognizable, one commissural, uniting the lateral lobes of the cerebellum, the other two decussating; of the latter the first set comes from the cerebellum and crosses to the opposite side, thence to pass onward to the cerebrum, while the second, which also decussates, probably goes chiefly to the cerebrum, but its fibres originate mainly in the pontile nuclei. These transverse fibres pass both before and behind the pyramidal bundles, those which are dorsad to the pyramid constituting a special tract known as the *trapezium*, or *corpus trapezoides,* many of the fibres of which pass around the superior olivary nucleus.

Cranial Nuclei and Nerve Roots.—Fig. 88 is a profile view of the right half of the oblongata and pons, supposed to be transparent, giving a fair idea of the course of the nerve roots and of the relative positions of the deep and superficial origins of the nerves. In several other illustrations the superficial or apparent origins of these nerves have been indicated, all being on the lateral and ventral aspects of the pons and oblongata: their

deep or true origins are in the preoblongata and postoblongata, and have been indicated when describing the floor of the fourth ventricle and its internal correlatives. It is important for diagnostic purposes to know not only the exact locations of the deep and apparent origins of these nerves, but also the manner in which the root fibres traverse both oblongata and pons to reach the ventral or lateral surfaces. In order to involve the cell nests of the cranial nerves, lesions must be situated in the extreme dorsal portions of the oblongatas, but root fibres may be attacked by lesions which affect any of the formations of the pons or oblongata. When treating of affections of the cranial nerves and of the substance of the pons and oblongata, this important subject will be more particularly discussed.

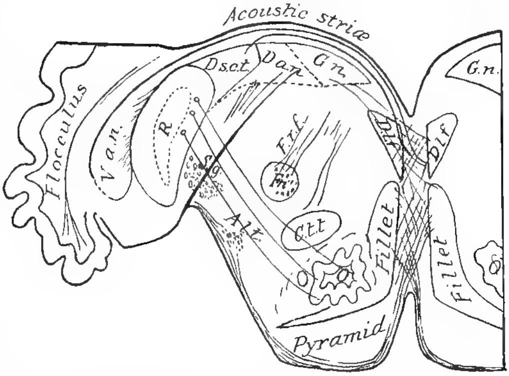
The Deep Origins of the Cranial Nerves.—It will be necessary here, however, to direct attention a little more fully to some points connected with the cranial nerves, and the other structures of the pons and oblongata, in order to understand their relations to each other and to certain tracts entering into the architecture and general physiology of the nervous system. Of the twelve pairs of cranial nerves, the olfactory issues from the forebrain; the optic from the interbrain; the common oculomotor and pathetic spring from the midbrain; the trigeminal, abducens or external oculomotor, facial, and acoustic from the hindbrain; and the glossopharyngeal, pneumogastric, accessory, and hypoglossal from the afterbrain. The deep as well as the superficial origins of the motor nerves are in the parts of the brain just indicated; but the nerves of common and of special sensibility originate in ganglia situated on their dorsal or posterior roots or tracts. The spinal and cranial nerves, both sensory and motor, are usually described as originating in regions of the spinal cord, oblongata, and other more cephalic structures at the base of the brain; but the intra-axial cell nests of the sensory nerves are best regarded as terminal nuclei for the peripheral elements of the sensory tract or path—as masses which link together the peripheral and the central elements of this tract. Their real origins are in the dorsal spinal ganglia, and in certain cranial ganglia, as the jugular, petrous, plexiform, and Gasserian. Axis cylinder prolongations from the ganglia of these sensory cranial nerves pass into the neuraxis, processes for the glossopharyngeal and the vagus going to the *ala cinerea* in the postoblongata, and those for the trigeminal nerve proceeding to its great terminal sensory nuclei in the *substantia gelatinosa.*

Ascending and Descending Roots of Sensory Cranial Nerves.—After entering the spinal cord the dorsal or posterior root fibres divide into T-shapes, or dichotomously, one branch running upward and the other downward, these branches forming the bulk of the posterior columns, and collaterals passing from them into the gray

substance, there to break up into nerve trees. The sensory subdivisions of the mixed cranial nerves—pneumogastric, glossopharyngeal, and trigeminal—follow the same course; that is, after entering the oblongata they bifurcate into ascending and descending branches. In various illustrations the location and course of the root fibres of the fifth nerve have been shown, as in Fig. 87, where they are seen closely related to the substantia gelatinosa of Rolando, or in the profile view, Fig. 88, where a long root is seen extending towards the spinal cord, and a shorter one towards the midbrain. This long root has until recently been regarded, and in most books is still designated, as the *ascending*; but this root is really a descending portion of the main sensory root, and with our present knowledge should be called descending or spinal. In a similar manner the *funiculus* or *fasciculus solitarius* (Fig. 87), or *solitary bundle of Meynert* (*respiratory bundle of Krause*), represents the descending roots of the sensory portions of the pneumogastric and glossopharyngeal nerves.

Nuclei and Root Fibres of the Acoustic Nerve.—The acoustic or auditory nerve, which arises superficially by lateral and mesal roots from the lower border of the pons, is subdivided into two parts. The lateral, dorsal, or posterior root is continuous with the *cochlear nerve*, and is the true nerve of hearing; while the mesal, ventral, or anterior root becomes the *vestibular nerve*, which is concerned especially with equilibration. The *spiral ganglion*, an arrangement of nerve cells also called *ganglion of Corti*, corresponds for the cochlear nerve to a posterior spinal ganglion, and the *ganglion of Scarpa* in the depths of the internal auditory canal, to the same for the vestibular division of the nerve. The oblongatal nuclei (Fig. 89) related to these nerves are usually regarded as three in number, the *ventral* or *accessory acoustic nucleus* or *auditory ganglion* (Bruce), on the outer side of the restiform body; the *dorsal* or *internal nucleus*, sometimes called the *chief nucleus*, lying beneath the floor of the fourth ventricle, and reaching the whole distance from the clavate

Fig. 89.

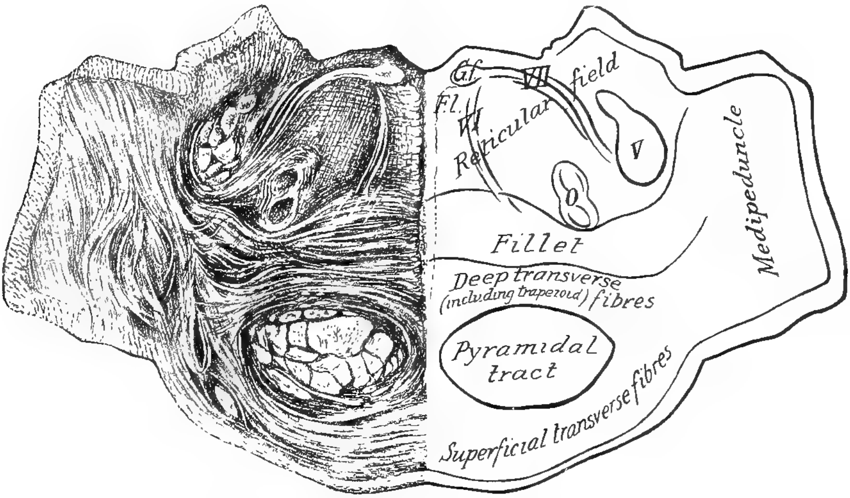


Transection showing the structure of the oblongata at the caudal border of the pons: *O*, inferior olive (olivary body); *R*, restis (restiform body); *S.g.*, substantia gelatinosa of Rolando; *D.s.c.t.*, direct cerebellar sensory tract, including Deiters's nucleus; *Alt*, anterior lateral tract (lateral medullary tract); *C.t.t.*, central tegmental tract; *D.l.f.*, dorsal or posterior longitudinal bundle; *V.a.n.*, ventral acoustic nucleus; *D.a.n.*, dorsal acoustic nucleus; *G.n.*, glossopharyngeal nucleus; *F.n.*, facial nucleus; *F.r.f.*, facial root fibres. (After Edinger.)

nucleus to the nucleus of the sixth nerve; and the so-called *external* or *superior nucleus* or *nucleus of Deiters*, on the inner side of the restiform body, although by some the acoustic connections of these two nuclei are regarded as doubtful. The cuneate nucleus has also relations of importance to the auditory nerve. In the courses taken by them after entering the neuraxis, the cochlear and vestibular branches of the auditory nerve show striking homologies with the spinal sensory and with other cranial sensory nerves; and the optic and olfactory nerves have similar homologies, all of which will be considered when the great sensory path is described.

The Nuclei of the Motor Cranial Nerves.—Through nearly the entire length of the oblongata, and seen therefore in numerous transections, is a large collection of cinerea, known as the *nucleus*

Fig. 90.



Transection through the pons in the region of the genufacialis: VI, abducens roots; Gf, facial knee (genufacialis); VII, emerging root of the facial nerve; V, descending (spinal) root of the trigeminal nerve; O, superior or minor olive. (Modified from Koelliker.)

ambiguus (Figs. 87 and 88) or *common nucleus*, which is practically a continuation of the ventral spinal horns, and from which arise the motor branches of the glossopharyngeal, pneumogastric, and spinal accessory nerves. The hypoglossus arises from a separate large nucleus. The nuclei of the seventh or facial nerve are situated in the reticular field, dorsad to the superior olive and somewhat deeply placed below the floor of the fourth ventricle. The positions and correlations of this nerve are shown in several illustrations. Its root fibres in their course form a curious double bend which is called the *genufacialis* (Fig. 90). Spitzka describes the facial bundle as curving over the abducens nidus and overlying it like a bent horseshoe over a ball. The nucleus of the sixth or abducens nerve is the ental

correlative of that portion of the terete fasciculus which lies just cephalad of the acoustic striæ. The motor nucleus of the trigeminal nerve, much smaller than its great terminal sensory nucleus, is situated a little more mesad and ventrad than the latter, beneath the cephalic portion of the metepicœlian floor. The nucleus of the fourth or pathetic nerve lies in front of the gray matter dorsad of the aqueduct, about midway between the pregeminum and the postgeminum, close to the lower end of the third nucleus. The cell nests of the third or common oculomotor nerve are in the ventral portion of the central cinerea of the aqueduct, close to the middle line, in several separate segments or groups of cells, which extend longitudinally from the postcommissure to a point nearly midway between the pregeminum and the postgeminum.

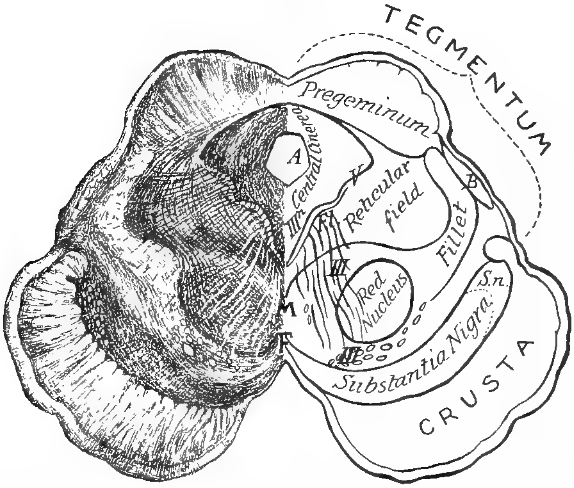
Special Ganglia and Nuclei in the Oblongata and Pons.—Besides the cell masses which are clearly related to the roots of the cranial nerves, other special nuclei and ganglionic masses are found in the oblongata and pons, some of known and some of unknown functions and relations. The largest of these is the *inferior olive* (*oliva, olivary body*), already described, which has near it two small nuclei, a *dorsal* or *outer* and a *mesal* or *inner olivary nucleus.* Other deposits or cell masses are the *superior olive,* the *central nucleus,* and the *lateral nucleus.* The superior olive (Fig. 90) is an ovoid mass which extends through the entire length of the pons in the ventral portion of the tegmentum, close to the trapezium, and has numerous connections with the nuclei of the cranial nerves and of the cerebellum, also with the postgeminum, and even with the spinal cord. The central nucleus is an accumulation of gray matter in the higher levels of the oblongata. The lateral nucleus, in the periphery of the oblongata, under the groove between the inferior olive and the restiform body, is composed of a ventral and dorsal group of cells, and probably represents the continuation of the lateral horns of the cord.

Nerve Tracts in the Oblongata and Pons.—The intricate nerve tracts in the oblongata and pons might perhaps be separated into numerous groups according to the directions taken by their constituent fibres, but, as shown in several illustrations in the near context, they are chiefly: 1, *longitudinal;* 2, *transverse*—including those which run an almost straight course and those which are more or less oblique in direction; and, 3, *arcuate* or *curving.* Two sets of fibres, which have been especially studied, cross the middle line near the pregeminum, one beneath the dorsal longitudinal bundle, called *Meynert's fountain-like tegmental decussation,* the other on the ventral side of the red nucleus, known as *Forel's ventral tegmental decussation.*

Longitudinal Tracts.—Some of the great nerve root tracts, as the descending branch of the trigeminus and the solitary bundle of Meynert, constitute longitudinal fibre tracts in the oblongata and

pons; but the longitudinal tracts of this region, as usually given, are: 1, the *pyramidal tract;* 2, the *lemniscus* or *fillet;* 3, the *lateral medullary tract;* 4, the *central tegmental tract;* 5, the *dorsal* or *posterior longitudinal bundle.* The pyramidal tract is the great cortico-muscular or motor pathway, and will be described in its entirety when considering the architecture of the nervous system. It occupies a large place in the crustal portion of the crura and pons, as shown in several figures; and in the oblongata it gets to the surface as the anterior pyramid. The lemniscus or fillet will be presently described in detail. The lateral medullary tract is a distinct strand or bundle in the most lateral portion of the reticular field, passing outward to the periphery of the oblongata. It lies just ventrad of the direct cerebellar tract, for which it has been mistaken.

FIG. 91.



Transection through the crus and pregeminum: *A*, iter or aqueduct; *B*, brachium conjunctivum; *Fl*, dorsal longitudinal bundle; *IIIn*, oculomotor nucleus; *III*, roots of the oculomotor cut longitudinally and transversely; *M*, Meynert's fountain-like tegmental crossing; *F*, Forel's ventral tegmental crossing; *Sn*, longitudinal bundle on the edge of the substantia nigra (intercalatum); *V*, descending root of fifth nerve. (After Koelliker.)

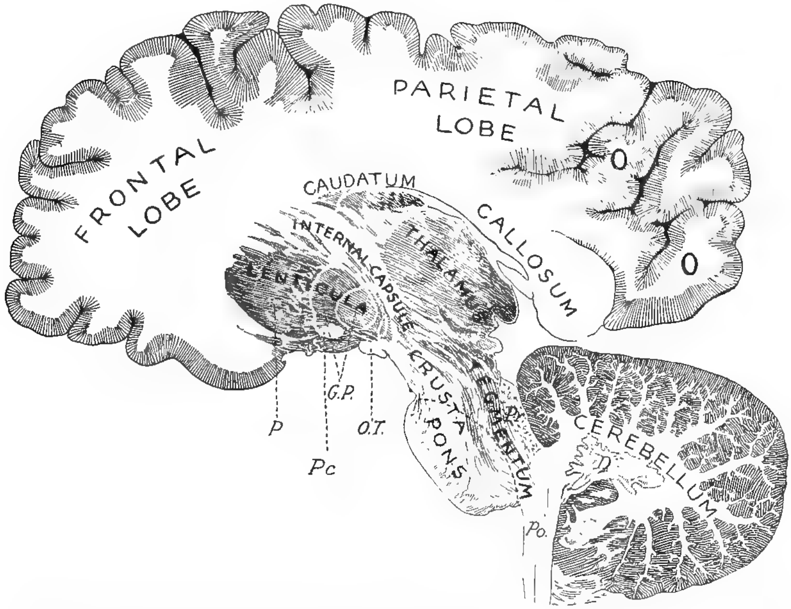
It contains Gowers's tract, and, according to Bruce, terminates in the lateral nuclei of the oblongata, in the lower pole of the superior olive, and by a special bundle which crosses the trapezium to enter the lateral fillet. Bechterew, Edinger, and others have demonstrated a *central tegmental tract*, beginning near the dorsal accessory nucleus and passing cephalad in the mid-tegmentum. In one of my cases of thalamic disease with involvement of the posterior limb of the internal capsule, this tract with the inferior olive was found to be markedly degenerated, showing that it probably reaches to the interbrain. According to Bechterew, it terminates in the lenticula. The dorsal longitudinal bundle already indicated

is a compact fasciculus which can be traced throughout the oblongata and pons as far cephalad as the nucleus of the third nerve.

Transverse Tracts.—The most important transverse fibres are the *superficial* and the *deep transverse fibres* of the pons, the latter including the *trapezium* or *trapezoid body.* These bundles are found respectively on the ventral and dorsal aspects of the pyramidal tracts.

Arcuate or Arciform Tracts.—Arcuate or curved fibres are to be seen in many transections of the oblongata or pons, and their arching course shows clearly in the planes of several of the transverse sections. These arcuate fibres are conveniently subdivided

FIG. 92.



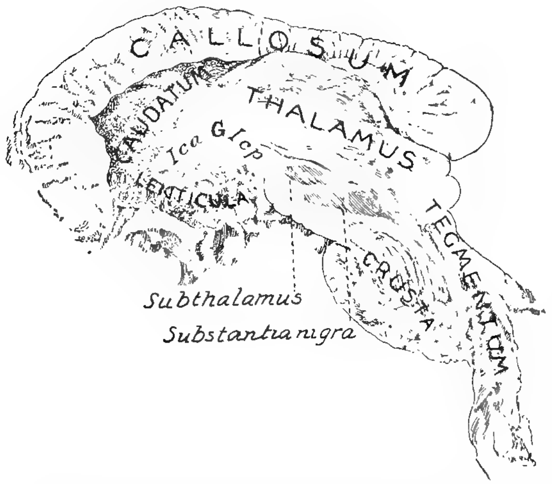
Longitudinal section of the entire brain through the cortex, alba, striata, thalamus, pons, oblongata, and cerebellum: *O*, occipital lobe; *P*, putamen; *G.P.*, pallidum (globus pallidus); *Pc*, precommissure; *O.T.*, optic tract; *Pr*, preoblongata; *Po*, postoblongata; *D*, dentatum (dentate nucleus).

into a *superficial* or *external* and a *deep* or *internal set.* Internal arcuate fibres constitute the decussation of the fillet; others from the clavate and cuneate nuclei pass through the dorsal longitudinal bundle and inferior olive to the fillet and restiform body of the opposite side; and still others pass from the inferior olive to the opposite restiform bodies. Many of these curving strands run from the nuclei of the motor cranial nerves, partly to the opposite dorsal longitudinal bundle and partly to this fasciculus on the same side. Others from the nuclei of sensory and sensorial nerves pass into the reticular field of either the opposite or the same side, probably into the central tegmental tract, which may serve to correlate the cell nests of the sensory cranial nerves, as the dorsal longitudinal fas-

ciculus does those of the motor. It is owing to the correlations which many of these tracts are intended to accomplish that their fibres take a curved or arciform course, some passing from longitudinal tracts to nuclei situated at different levels and in different longitudinal planes; some leaving nuclei and decussating to enter important longitudinal tracts either ascending or descending; some serving to connect ganglionic masses with others widely separated in the midbrain, hindbrain, or afterbrain.

The Crusta and the Tegmentum.—The crura and pons are conveniently subdivided into a ventral or anterior portion, the *crusta*, and a dorsal or posterior, the *tegmentum*. The former is also known as the *pes* or *fuss*, the latter term being commonly used by the Germans. These subdivisions in the cerebrum pass among the basal ganglia—the crusta in the main *pyramidal tract*, which comes from the motor cortex, and the tegmentum as the tegmental radiation, which probably goes chiefly to the limbic lobe and the postparietal region. Below the pons the crusta and tegmentum blend with less definition with the ventral and dorsal structures of the oblongata. They appear most distinctly as separate formations in a transection through the crura just above the pons. The tegmentum is much darker in appearance because it has in it many nerve cells, which in some places are arranged in well defined nests, deposits, or ganglia. At the cephalic extremity of the crura, close to the pulvinar, the crusta contains fibres from the motor cortex and from both the frontal lobe and the temporal lobe to the pons. Fig. 92 represents a longitudinal section through the entire right half of the brain, close to the raphe of the callosum, showing the relations of the crusta and tegmentum to the more cephalic structures and also to the oblongata and cerebellum. Several of the subthalamic structures are shown in Fig. 93, the section being made in a slightly different plane.

Fig. 93.



Longitudinal section through the basal ganglia, subthalamus, substantia nigra (intercalatum), pons, and oblongata: *Ica*, anterior limb of internal capsule; *Icp*, posterior limb of internal capsule; *G*, genu or knee of internal capsule.

Intercalatum, or Substantia Nigra.—Sometimes the tegmentum and crusta are described as separated by the *substantia nigra* or *locus niger*, names which have been given because of the dark color of the mass, which is due to pigmented cells. Spitzka has proposed the name *intercalatum*, referring to its constant intercalation between the tegmentum and the crusta. The intercalatum is a collection of nerve cells and fibres the functions of which are as yet unknown. It can be demonstrated from the subthalamic region downward to the midbrain.

The Lemniscus or Fillet.—The *lemniscus* or *fillet* (Fig. 94) is best studied by a series of transections beginning at the crura and passing downward to the junction of the oblongata and the cord. It is divided into three parts: a division which probably goes to the parietal and limbic cortex and hence has been called the *cortex lemniscus tract;* another subdivision, which goes to the pregeminum and to the thalamus; and a lower *lateral division*, which passes to the postgeminum. It is a part of the great sensory tract, which is in the most ventral portion of the tegmentum. On its way brainward it gathers in the fibres which come from or go to the cell nests of those cranial nerves which have sensory functions.

FIG. 94.

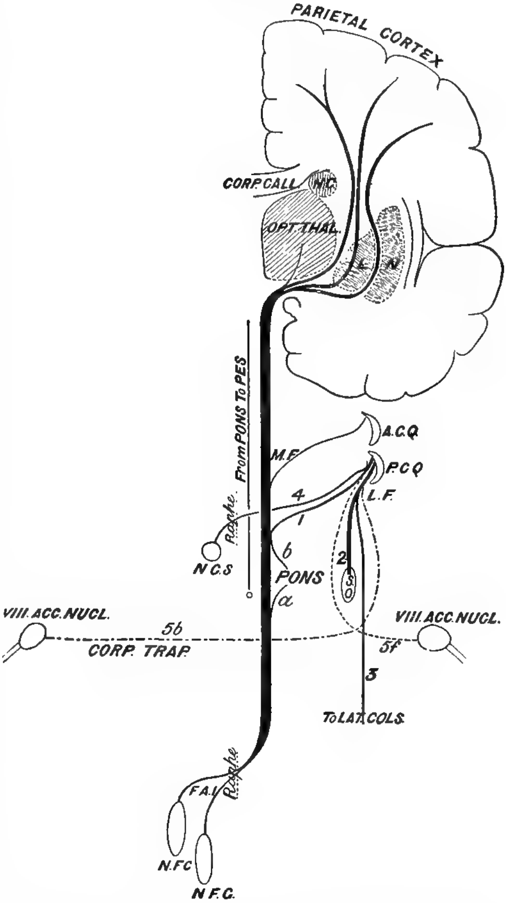
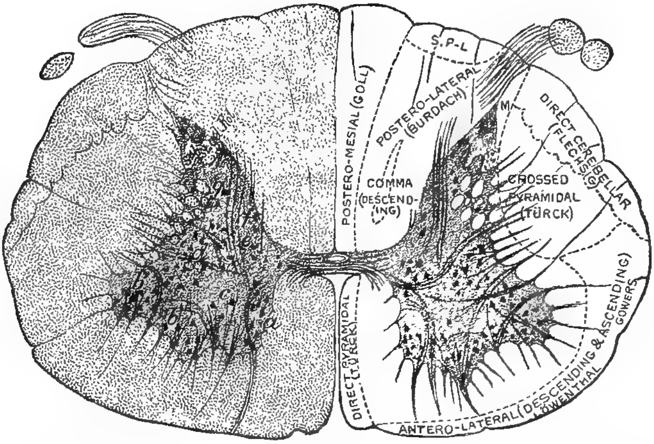


Diagram to show the constituents of the fillet: *N.F.G.*, nucleus funiculi gracilis (gracile or clavate nucleus); *N.F.C.*, nucleus funiculi cuneati (cuneate nucleus); *F.A.I.*, internal arcuate fibres crossing the raphe; *a*, fibres to pons; *b*, fibres from pons; *M.F.*, median fillet; *L.F.*, lateral fillet; *P.C.Q.*, postgeminum; *A.C.Q.*, pregeminum; *Opt. Thal.*, thalamus; *L.N.*, lenticula; *S.O.*, superior olive; *N.C.S.*, nucleus centralis superior. Lateral fillet: 1, fibres from median fillet to postgeminum; 2, fibres from superior olive to postgeminum; 3, lateral medullary tract; 4, fibres from nucleus centralis superior to lateral fillet; *VIII.Acc.Nucl.*, accessory nucleus of auditory nerve; 5*f*, Flechsig's tract from accessory nucleus to lateral fillet by way of trapezium (corpus trapezoides); 5*b*, Baginsky's tract from accessory nucleus of opposite side to lateral fillet by trapezium. (Bruce.)

Chief Subdivisions of the Spinal Cord.—The appearance, dimensions, and some of the relations of the spinal cord to its nerves and to other parts have already been briefly described, and, as has been stated, transections of the spinal cord reveal a peculiar arrange-

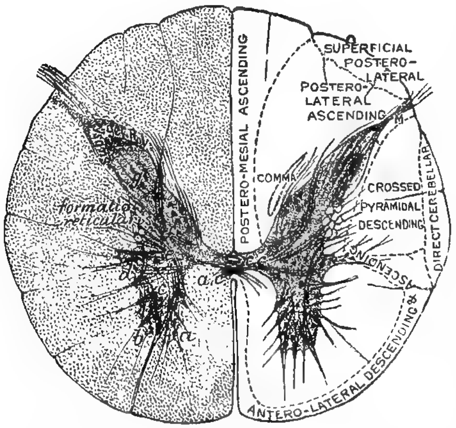
FIG. 95.



Transection of spinal cord in lower cervical region. (Schäfer, in Quain's Anatomy.)

ment of gray and white matter differing somewhat in bulk and appearance at different heights. It is necessary at this point to study the main divisions of the spinal cord, as revealed by these sections, in order to understand its relations to other portions of the nervous system. In Figs. 95, 96, and 97 are shown semi-diagrammatic views of transections in the lower cervical, thoracic, and lumbar regions. The spinal cord is composed of symmetrical halves, separated dorsally by a septum and ventrally by a fissure. Its gray matter forms an H-like mass, the bar of the H being composed of a gray and a white or whitish commissure. The bulk of the gray matter is divided into a ventral or anterior horn and a dorsal or posterior horn, the former broader and more massive than the latter. The ventral horn is placed at a considerable distance from the periphery of the cord;

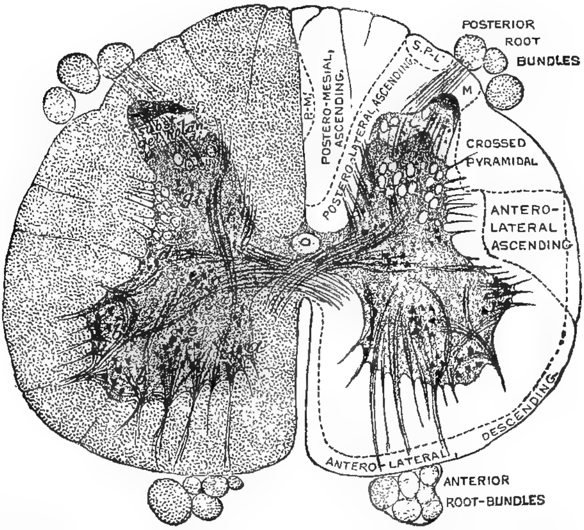
FIG. 96.



Transection of spinal cord in lower thoracic or dorsal region. (Schäfer, in Quain's Anatomy.)

while the dorsal horn reaches by its pointed extremity to the surface; and near its tip this horn is capped by a semitransparent substance, the *substantia gelatinosa of Rolando.* The gray substance intermediate between the two horns has been named by Gowers the *intermediate gray substance.* In the thoracic region is a projection about the position of the intermediate gray substance, sometimes spoken of as the *lateral horn,* and sometimes as the *intermediate process of Gowers,* or the *intermediolateral tract* of Lockhart Clarke. What is known as *Clarke's column* or the *posterovesicular column of Clarke* is a column of nerve cells (*f*) grouped in the inner portion of the neck of the dorsal horn, principally between the eighth cervical and third lumbar segments. Nerve cells are arranged in groups in the sub-

FIG. 97.

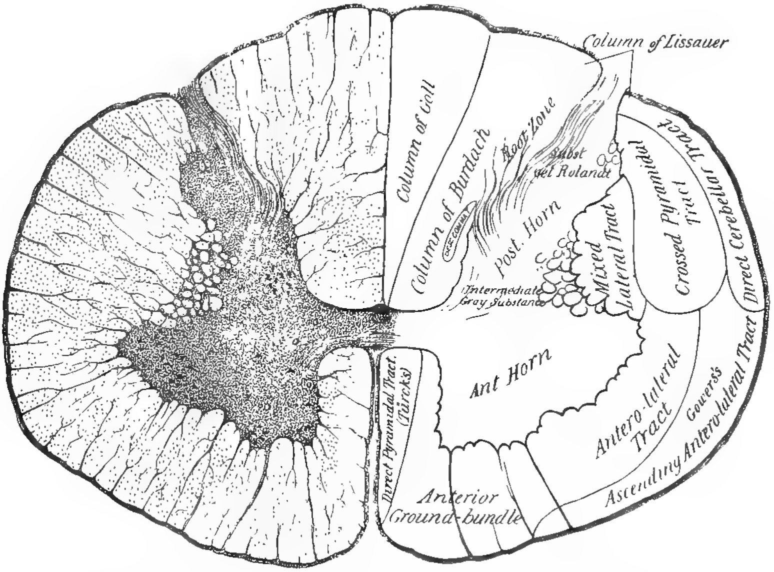


Section of spinal cord in lower lumbar region. (Schäfer, in Quain's Anatomy.)

divisions of the gray matter at all levels, and these superimposed groups form *cell columns* or longitudinal tracts of cells. The best defined of these groups are indicated in the illustrations at *a*, *b*, *b'*, *c*, *d*, *e*, *f*, and *g*, in Figs. 95, 96, and 97. A general division of the white matter of the spinal cord is made into a *ventral* or *anterior*, a *dorsal* or *posterior*, and a *lateral region*, and these have important subdivisions which have been determined by anatomical, pathological, and physiological investigations. They need in this connection to be little more than named: when the diseases of the spinal cord are considered they will demand more attention. The columns or tracts of the ventral region are *Türck's direct pyramidal tract*, descending and motor; and the *ventral* or *anterior ground bundle* or *anterior root zone*, which is crossed by many nerve fibres from the anterior

horns. The two most prominent tracts of the dorsal or posterior region are the *posteromedian tract*, or *column of Goll*, and the *posterolateral tract*, or *column of Burdach*, which latter is in part a root zone. At the end of the posterior horn is a small but well defined bundle of nerve fibres, known as the *zone*, *tract*, or *column of Lissauer*, described by Lissauer, and also by Spitzka, Bechterew, and others. These tracts are almost exclusively ascending and sensory, but not far from the centre of the column of Burdach is a narrow zone, which has been described as the *descending comma tract*, in which fibres are constantly observed to degenerate downward. Of the tracts of the lateral columns some are ascending and some descending; some are sensory or afferent paths, and some are motor

FIG. 98.



Transection of cervical spinal cord, showing its chief subdivisions.

or efferent, facts which are in the main indicated in the diagrams. The *crossed pyramidal tract* is motor, and of course descending. The dorsolateral or *direct cerebellar tract* or *bundle*, sometimes called the *direct lateral cerebellar tract of Flechsig*, is ascending and sensory. *Gowers's tract* is ascending and sensory, and *Lowenthal's* is descending or motor, and is connected with the cerebellar cortex. In some diagrams the separation of these into two divisions is not made, because the precise limitations of each have not been fully made out. It is probable that in the *mixed lateral tract* the dorsal or posterior division contains sensory, and the ventral or anterior motor fibres. Fig. 98, drawn by Dr. J. C. McConnell, from a study of sections and of various diagrams, shows some parts just described, but not indicated in the other illustrations.

Vertical Extension of the Spinal Tracts.—The columns of Goll and Burdach are distinctly separated in the cervical region and less so in the lower thoracic, but in the lumbar portions of the cord the distinction cannot be clearly made. The descending comma tract is best defined in the upper thoracic and lower cervical regions. The zone of Lissauer is present at all levels. The direct cerebellar tract reaches only from the level of the first lumbar nerve upward. The crossed pyramidal tract extends through the length of the cord; so also do the anterolateral column and lateral limiting layer, both of which are largely composed of fibres which have only a short course within the cord. The anterolateral ascending tract of Gowers probably begins at a lower level than the direct cerebellar tract. Türck's column is usually considered as present only in the cervical and upper thoracic regions, but it has been traced to a much lower level. In considering a level below the upper thoracic region, the diagnostician would not usually take into account the column of Türck. The direct cerebellar column would also in large measure not be regarded in lesions of the lumbosacral cord and the cauda equina, even although in the lesions of the lower lumbar region the tract of Gowers might be involved. On the other hand, the crossed pyramidal tract and anterolateral columns would receive consideration at any and all heights.

The Structural Elements of the Spinal Cord.—The structural elements of the spinal cord, and their relations to each other, to the periphery of the body, and to the higher levels of the nervous system, have been elucidated by the labors of Golgi, Ramón y Cajal, Koelliker, Edinger, Van Gehuchten, and Lenhossék. Fibres from the dorsal or posterior ganglia enter the white substance, and, dividing into ascending and descending branches, give off, at right angles to their course, twigs which pass into the gray matter, there breaking into tufts or ending in free fibrils. These may be in contact with the nerve cells, but are not structurally continuous with them. Cells of the ventral or anterior horns send out axis cylinder processes, which become true nerve fibres of the motor nerves or of the ventral or lateral columns of the cord. That fibres originate in the cells of the gray substance has been proved by actually tracing their courses, and also by experiments like those of Singer and Münzer, who found that destruction of the lumbar cinerea was followed by extensive degeneration of fibres of the anterolateral columns. The gray substance of the spinal cord, as of the entire neuraxis, is constituted chiefly of cells, or rather of cell bodies, between which ramify innumerable collateral and terminal fibres. Golgi discovered that some of these cells have short and some long axis cylinders; and to those with short processes the name *Golgi's commissural cells* has been given. They have been observed especially in the dorsal horns. Van Gehuchten divides the cells of the spinal

cord into *root cells* and *column cells*, and the former into *cells of the anterior or ventral* and the latter into *cells of the posterior or dorsal*

FIG. 99.

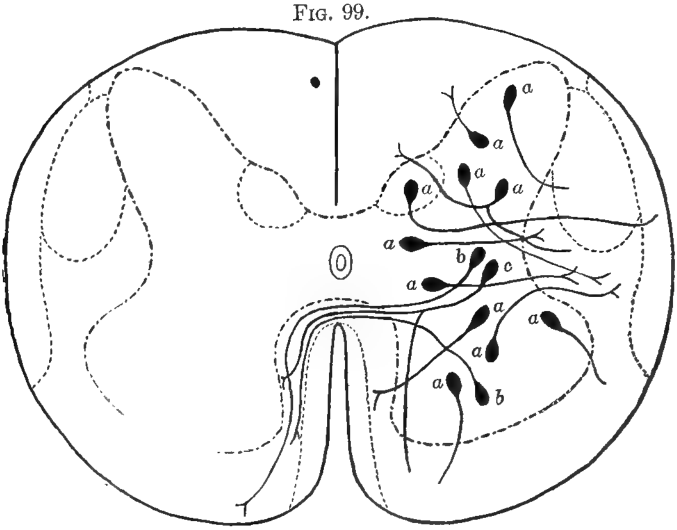
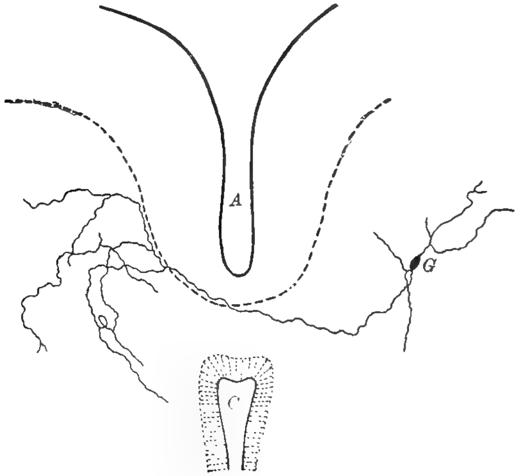


Diagram showing *a*, tautomeral; *b*, heteromeral; and *c*, hecatomeral cells.

roots. The column cells have been subdivided by him into three groups: (1) cells the axis cylinders of which pass into the white

FIG. 100.



Golgi's commissural cell (from the cervical region of an eighteen millimetre long human embryo): *G*, Golgi's commissural cell; *A*, anterior longitudinal fissure; *C*, central canal. (Lenhossék.)

substance of the side on which they originate, or *tautomeral cells*; (2) cells the axis cylinders of which proceed by way of the ventral

or anterior commissure to the white substance of the opposite side, or *heteromeral cells;* and (3) cells the axis cylinder processes of which bifurcate in the gray substance, one branch passing into the white substance of the same side, and the others by the anterior commissure to the anterolateral columns of the opposite side, or *hecatomeral cells.*[1] These are shown diagrammatically in Fig. 99. Fig. 100 represents a so-called Golgi's commissural cell,—here a hecatomeral cell whose axis cylinder process passes by way of the anterior com-

FIG. 101.

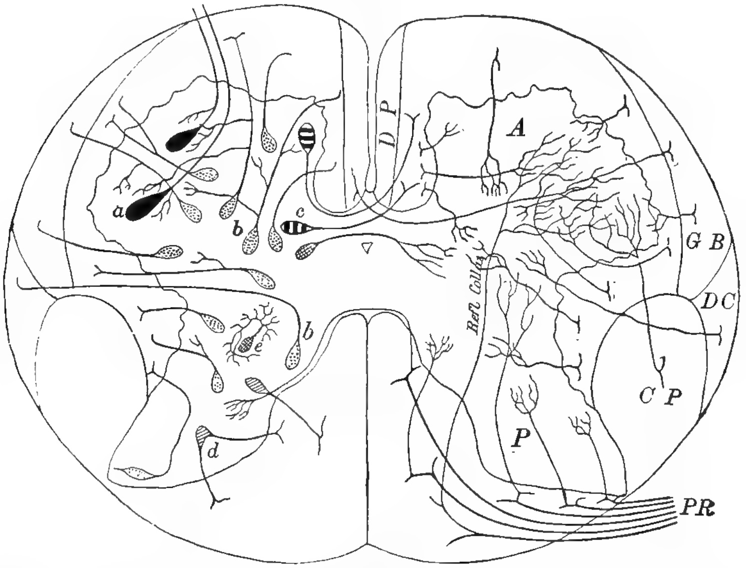


Diagram illustrating the chief cellular elements of the spinal cord, and the probable relations between the cells and the fibres and the principal tracts; the left half of the figure exhibits the communications of the several varieties of nerve cells: *A*, *P*, ventral or anterior and dorsal or posterior horns; *PR*, posterior root bundles; *DP*, direct pyramidal tract; *CP*, crossed pyramidal tract; *DC*, direct cerebellar tract; *GB*, Gowers's tract; *a*, motor cells passing directly into fibres of ventral roots; *b*, various cells of the anterolateral column; some give off collateral branches of remarkable size; *c*, commissural (heteromeral) cells; *d*, cells to dorsal column (tautomeral); *e*, Golgi cells of dorsal horn. The right half of the diagram shows the communications established by means of the collateral fibres. (Piersol, after Lenhossék.)

missure to the opposite side of the cord. Fig. 101 shows the chief cellular elements of the spinal cord and their probable relations to each other and to the horns and principal columns. Ventral motor root cells are shown at *a*, and dorsal root bundles at *PR*; at *b* are cells of the anterolateral columns with fibres going only to the side on which the cells originate; at *d* cells going to the posterior columns of the same side; at *c* commissural cells of the heteromeral variety. Hecatomeral cells are not shown in the figure. At *e* are

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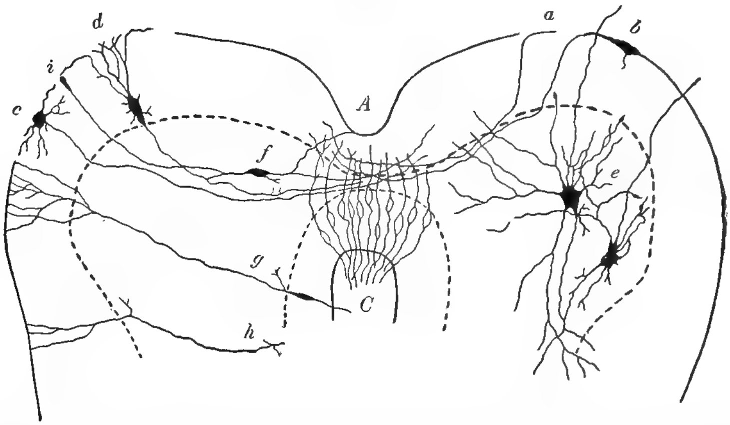
[1] These words are derived from the Greek τὸ αὐτὸ μέρος, the same part or *side;* ἕτερον μέρος, the other side; and ἑκάτερον μέρος, both sides.

seen the cells with short processes peculiar to the dorsal horns. The diagram also represents on one side the methods of communication by means of collaterals, and on the other by different forms of nerve cells. At one point is seen a long reflected collateral. Collaterals of the dorsal portion of the lateral columns cross by way of the dorsal or posterior commissure; and protoplasmic processes of the cells of the dorsal horns are also said to decussate.

Lenhossék's Commissural Cell.—Lenhossék has described a special form of commissural cell (Fig. 102), which, passing from the ventral horn, leaves the white substance between the ventral and lateral columns, and sometimes reaches the surface of the spinal cord, where it becomes flattened out like a scale through loss of its dendritic processes. On transverse section, as in *a*, these cells

Fig. 102.



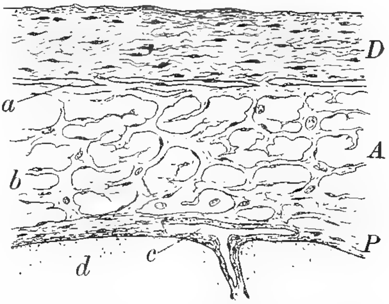
Commissural cells reaching the circumference of the spinal cord (from a nine days' old chicken); *a*, *b*, *c*, *d*, various forms of these commissural cells; *e*, motor cell; *f*, commissural cell with peculiar dendritic processes; *g*, ependyma cell; *h*, glia cell; *A*, anterior ventral longitudinal fissure; *C*, central canal. (Lenhossék.)

appear as thin lines which can hardly be recognized as cells; in *b* the cell body is coarser, more defined, and has a short process passing into the white substance, and giving also an unbranched protoplasmic process; in *c* the cell approaches closely the common type, having many dendritic processes, except at its free border; while at *d* is seen a cell much less displaced, and in the innermost part of the anterior column the cell is spindle shaped, its axis being directed towards the periphery. The real dendritic processes spring from the outer, the axis cylinder prolongations from the inner point. Near the anterior commissure one of these spindle shaped commissural cells is seen lying obliquely at *f*, and this differs from the ordinary type, having on its point a coarse protoplasmic process which passes through the gray and white substance to the surface of the cord, to end there in a large bulb. Glia cells are also shown.

MEMBRANES, BLOODVESSELS, AND LYMPHATICS OF THE NERVOUS SYSTEM.

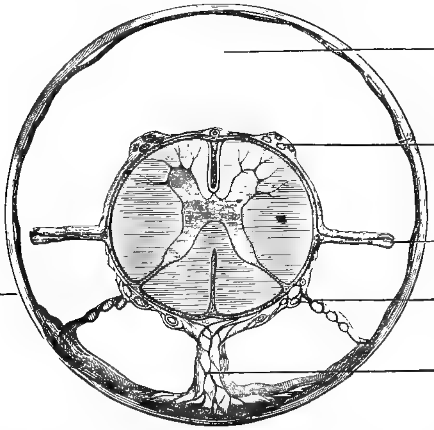
Envelopes of the Brain and Spinal Cord.—The brain is invested by three membranes, called respectively the *dura*, the *pia*, and the *arachnoid*, although the last two are often so united as practically to form one membrane. The dura lines the interior of the skull and also supports and protects the brain. A great process of the dura, called the *falx*, passes between the two hemispheres of the cerebrum into the great longitudinal fissure; another, the *tentorium*, separates the cerebrum and the cerebellum, and the *falcula* or cerebellar falx dips between the two cerebellar hemispheres. The pia closely invests the brain, everywhere following the contours of its surface and dipping into its fissures, even passing by way of the great transverse fissure into the ventricles. The arachnoid closely envelops the pia, but does not pass into the fissures, except those, like the longitudinal, which contain processes of the dura. In Fig. 103 are shown the microscopical appearances of a section of the membranes from the brain of a child. The

Fig. 103.



Section of membranes from brain of child: *D, A, P*, respectively the dura, the arachnoid, and the pia; *a*, subdural space; *b*, meshes of subarachnoidean space; *c*, bloodvessel within the pia sending branch into cerebral cortex, *d*. (Piersol.)

Fig. 104.



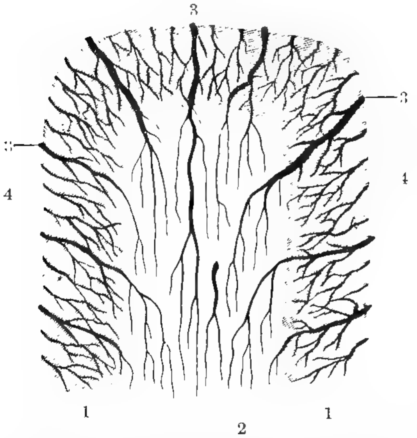
Transection of the spinal cord and its membranes. (Morris's Anatomy, from Key and Retzius.)

spinal cord, like the brain, is invested with three membranes. The dura is a loose sheath around the cord and the cauda equina.

Externally it is loosely connected with the periosteum and ligaments of the spinal canal, and a narrow subdural space separates it from the pia-arachnoid. Processes of the pia, called the ligamentum denticulatum, pass across the subdural space between the two membranes. The spinal arachnoid is a thin membrane continuous with the cerebral arachnoid. The spinal pia has an outer and an inner layer, the latter being continuous with the pia of the brain. Fig. 104 shows the spinal cord and its membranes in transection.

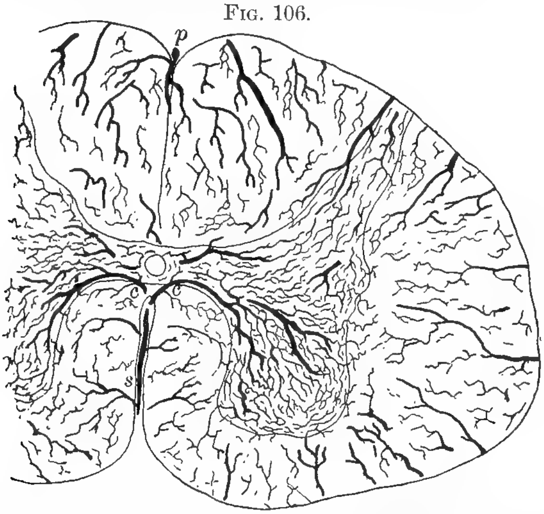
Bloodvessels of the Central Nervous System.—The functions of nervous tissue being in a high degree specialized, and requiring abundant blood, and yet a supply which often changes abruptly, its vessels are adapted to do their work with the greatest facility and with the least danger of rupture. Both in the brain and in the spinal cord are two vascular systems, one cortical or circumferential and the other central. From the cerebral and cerebellar arteries certain branches dip into the central region of the brain; other distinct branches pass to the brain cortex. The branches from the anterior, middle, and posterior cerebral arteries which pass to the various convolutions of the brain, in their course send off innumerable small vessels which anastomose and then go down through the cortex to terminate almost straight in the alba or centrum ovale. The terminations of the central and of the cortical vessels are near to each other, but they do not communicate. Even between different cortical systems but little communication exists. Some of the cortical vessels are short and some are long, and the latter alone penetrate to a considerable depth. A section across a cerebral convolution (Fig. 105) exhibits the arrangement of cortical arteries. The blood supply to the spinal cord is by numerous vessels, and is ventral and dorsal, the central supply being chiefly ventral, by way of the ventral or anterior mesal fissure from the anterior spinal artery. Its arterial supply is well shown in Fig. 106. The veins divide and subdivide and are distributed to different portions of the nervous system in much the same manner as the arteries. Like the arteries, they subdivide dichotomously, that is, by bisecting in pairs. Within both the

FIG. 105.



Section across a cerebral convolution, exhibiting the arterial arrangement: 1, the gray cortex; 2, interior white matter; 3, medullary arteries penetrating the cortex to the white matter; 4, cortical arteries distributed to the cortex. (Leidy, after Duret.)

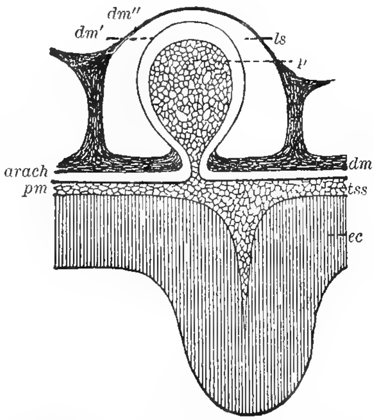
FIG. 106.



Section of injected spinal cord of child: *s*, sulcal branch of anterior spinal artery occupying ventral or anterior median fissure; *c*, *c*, sulco-commissural vessels from sulcal artery passing to gray matter to form dense network; *p*, posterior spinal artery, sending off twigs to white matter; around margin of cord numerous peripheral vessels enter white substance to form open network. (Piersol.)

cranium and the spinal canal the arrangement of veins and sinuses is such as to guard against serious accidents from disturbances of the circulation. Communication between the different sinuses of the brain is free; it is also comparatively free between the intracranial and the extracranial sinuses and veins, and between these and the spinal sinus system. The cerebral veins and sinuses have no valves.

FIG. 107.



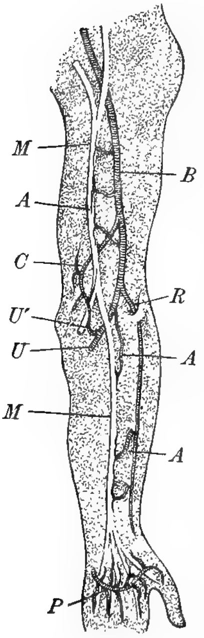
Diagrammatic figure showing the course of a Pacchionian granulation with the lac sanguin of the dura: *p*, villosity of the arachnoid; *dm*, dura; *dm'*, deep fold or lining of the dura; *dm''*, superior lining or fold of the dura; *ls*, lac sanguin; *arach*, arachnoid; *tss*, subarachnoid tissue; *pm*, pia; *ec*, gray matter of the brain. (Van Gehuchten, after Schwalbe.)

Venous Lakes or Lacs Sanguins.—Besides the venous sinuses of the brain, small irregular cavities are found on each side of the superior longitudinal sinus. These were discovered in 1853 by Faivre, and were described in 1868 by Trolard, who called them *lacs sanguins*. They are intradural cavities which communicate with the superior longitudinal sinus, with the meningeal veins, and with the cerebral veins. Trolard considered them reservoirs to receive momentarily the overflow of venous blood when the cranial

circulation was congested. These little cavities or lakes are found just at the level where the small granular white masses on the external surfaces of the dura, known as Pacchionian granulations, are most abundant (Van Gehuchten). Fig. 107 shows a Pacchionian granulation with the lac sanguin.

Arterial Supply of Nerves.—As an artery approaches the nerve sheath it divides into pairs, one branch going upward and one downward, sometimes on the outside and sometimes on the inner side of the sheath or epineurium. These branches divide and subdivide again and again. They pierce the perineurium at an oblique angle, and when they reach the interior of the nerve bundles they break up into fine capillary networks. The arrangement of vessels in the nerves, as in the brain and in the cord, is such as to prevent bad effects from sudden changes in circulation. Fig. 108 shows the distribution of the arteries to the median nerve.

FIG. 108.



Arteries of the median nerve: *A*, *A*, *A*, branches to the nerves; *B*, brachial artery; *C*, anastomotica; *R*, radial; *U*, ulnar; *U′*, anterior ulnar recurrent; *P*, superficial palmar arch; *M*, median nerve. (Quénu and Lejars.)

Lymphatics of the Nervous System.—As the part played by the lymphatics in nervous disease is one of increasing importance, the student and physician should have some knowledge of the arrangement and distribution of this system in the neuraxis and nerves. The interior of the skull is rich in lymph tracts, which, according to Rauber, are divided into six groups: 1, adventitious, perivascular lymphatic sheaths which surround the bloodvessels down to the capillaries, and open on the exterior of the brain into the subarachnoid space; 2, epicerebral lymph tracts, which lie between the pia and the surface of the brain; 3, the interpial lymph space, between the two layers of the pia; 4, the subarachnoid lymph space, between the pia and the arachnoid; 5, the subdural lymph space, an extensive although capillary lymph cleft or slit, between the dura and the arachnoid; 6, the epidural lymph space, a system of lymph clefts or slits on the outer surface of the dura. The choroid plexuses have rich lymphatic networks which belong to the system of interpial lymph clefts. The same lymphatic clefts occur in the spinal cord. A special epidural lymph space of the spinal cord has not been proved, but probably exists.

Lymphatics of the Interior of the Brain.—Rossbach and Sehrwald described lymphatics in the brain substance; but their presence in the brain has been denied by Sappey and others, and the evidence indicates that they do not exist as independent vessels in

the tissues of the nervous system, but that, as above described, around the bloodvessels of the brain and cord are loose adventitial lymph spaces which open into the subarachnoid space. The perivascular spaces of His are supposed by Retzius to be artefacts. According to Bevan Lewis, a system of lymph connective elements or scavenger cells with numerous prolongations drains the areas between the minute branching vessels.

Subarachnoid Lymph Spaces.—The subarachnoid lymph space extends everywhere beneath the arachnoid, and has numerous subdivisions of greater or less size, these in some positions forming *cisterns* or *reservoirs*. The largest, situated between the oblongata and cerebellum and adjacent portions of the cranium, the *posterior subarachnoid space* of the older anatomists, has been described as the *cisterna magna cerebello-medullaris* by Key and Retzius, and as the *postcisterna* by Wilder. The intracrural cistern or *ventricisterna* of Wilder — between the crura, pons, tuber, infundibulum, and hypophysis—has been usually described as the *anterior subarachnoid space.* Subarachnoid spaces, large and small, communicate with one another, and with the neurocœle, or connected ventricular cavities, at various places, as by the foramen of Magendie, by a cleft in the pia along the descending horn, and by lateral apertures in the fourth ventricle. That the ventricles of the brain constituted a great lymph cavity was correctly asserted by Willis in the seventeenth century. The central spinal canal and the brain ventricles are to be regarded as primitive lymph spaces which at a certain stage of fetal development get into direct communication with the subarachnoid lymph spaces. The discharge ways of the lymph of the brain, the spinal cord, and its membranes are through all the openings in the skull and spinal column: even microscopic bloodvessels and canals take part in this process. Of the large cranial openings, the carotid canal, the jugular foramen, and the tract of the vertebral artery deserve especial attention. The existence of lymphatics in the dura is by some regarded as doubtful, although it shows numerous clefts, probably lymph channels.

Lymph Vessels to the Nerves.—The subdural and subarachnoid lymph spaces are continuous with the spinal lymph spaces outward and along the nerves, and in an especially complete manner with the olfactory and auditory nerves. Along the nerves in general the subdural and subarachnoid spaces merge, the arachnoid disappearing. Flatau believes that in the rabbit and cat it is possible to inject lymphatics of the nose from the subarachnoid space, and holds that a number of "reflex nasal neuroses," so called, may be due to a derangement of the lymphatic circulation in these channels. The injection follows the olfactory nerve fibrils in perineural channels. Key and Retzius had already made experiments on this point, although they were not free from error.

ARCHITECTURE AND GENERAL PHYSIOLOGY.

Encephalospinal Conducting Paths.—While some of the views now held differ somewhat in their details from those indicated in Fig. 109, this diagram serves an admirable purpose in showing some

FIG. 109.

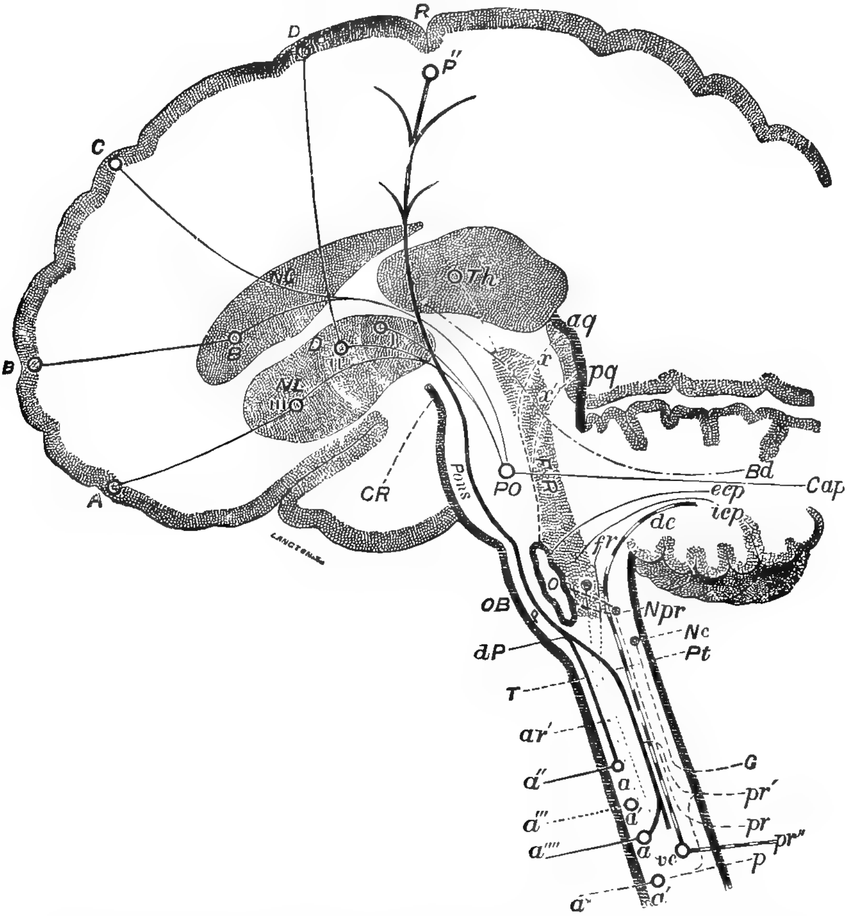


Diagram showing the paths between the gray masses of the spinal cord and brain: *R*, central fissure or fissure of Rolando; *P''*, *P*, *T*, and *Pt*, course of the pyramidal tract from the central convolutions to the ventral or anterior horns (*a*, *a'*); *I*, *II*, *III*, first, second, and third portions of the lenticula (lenticular nucleus) (*NL*); *NC*, caudatum; *Th*, thalamus; *D*, *C*, *B*, *A*, points from which fibres issue connecting the cortex of the brain and basal ganglia, and also the gray substance of the pons (*PO*); *Bd*, fibres connecting the cerebellum and thalamus; *Cap*, fibres connecting the cerebellum and gray substance of the pons; *aq*, pregeminum, and *pq*, postgeminum; *x*, upper, and *x'*, lower fibres connecting the olive and the quadrigeminum; *FR*, formatio reticularis of the oblongata, formed by fibres from the thalamus (*Th*), the internal division of the postpeduncle (*icp*), from the spinal cord (*fr*, *ar*, and *ar'*), and probably also from the clavate nucleus (*Nc*); *O*, oliva (inferior olive); *ecp*, fibres of the restiform bodies connecting the olivary bodies and cerebellum; other fibres connect it with the cuneate (*Npr*) and clavate (*Nc*) nuclei; *dp*, decussation of the pyramids; *pr'*, fibres of the dorsal or posterior roots which pass upward and downward into the gray substance, and pursue only a short course; *a*, *a'*, *a''*, *a'''*, ventral or anterior roots; *p*, *pr*, *pr''*, *G*, fibres of the dorsal or posterior roots. (After Flechsig.)

of the most important encephalospinal connecting paths. The main centres and areas shown are (1) the cortex of the brain, the central fissure indicated at *R*; (2) the ganglia at the base of the brain, namely, the lenticula, the caudatum, and the thalamus; (3) the quadrigeminal body; (4) the formatio reticularis; (5) pontile nuclei; (6) the olivary body; (7) a section of the cerebellum; (8) nuclei and nerve roots of the spinal cord. The most important conducting path here shown is the pyramidal tract. The striata are connected with the brain cortex, and with the nuclei of the pons, and the cortex has also direct connections with these nuclei. The quadrigeminal body is united by tracts with the inferior olive, and also, although this is not represented, with the optic and auditory tracts. The cerebellum has connections with the pontile nuclei, with the striata and thalamus, with the cortex, with the inferior olive, and with the spinal cord. Fibres of the posterior root are seen passing upward and downward in the spinal gray substance. At *Nc* and *Npr* are indicated the *clavate nucleus* and the *cuneate nucleus*. Schemes of the nervous system are given in Fig. 110, by Schäfer, which shows the relations of some of the principal cells and fibres of the nervous system to one another, and in Figs. 111 and 112, which are schemes by Van Gehuchten of the sensory and pyramidal tracts or paths.

The Great Sensory Path.—The sensory nerves sooner or later send their processes into the great sensory tract which eventually reaches the thalamus and cortex of the opposite side. The spinal peripheral sensory nerve cells originate in the dorsal or posterior ganglia and send their axis cylinders into the gray substance of the cord. Early in embryonic life these cells are bipolar or dineuric; and in some lower animals they so continue; but in man they apparently become unipolar by the fusion of two prolongations which were primarily independent. A long prolongation becomes the fundamental portion of the peripheral nerve fibre, passing to the end organ or terminating by free branches; while a shorter and finer prolongation becomes the dorsal spinal root, which penetrates the posterior column, there bifurcating, one branch ascending and another descending, to constitute the bulk of the white substance of this column. These branches send out numerous collaterals. Ascending fibres decussate in two ways: (1) by the crossing of separate cell processes in successive segments of the spinal cord, and (2) by a crossing in block at the upper sensory decussation, or decussation of the fillet, as shown in Fig. 111. The processes which cross at all levels probably pass into the ascending anterolateral tract of Gowers, and are supposed to be the paths for the pain and temperature senses; while the long fibres of the direct path which ascend in the columns of Goll and Burdach are supposed to transmit tactile impressions. Certain short processes do not enter either of these tracts, but make up the spinal reflex paths. The ascending sensory tract is augmented

Fig. 110.

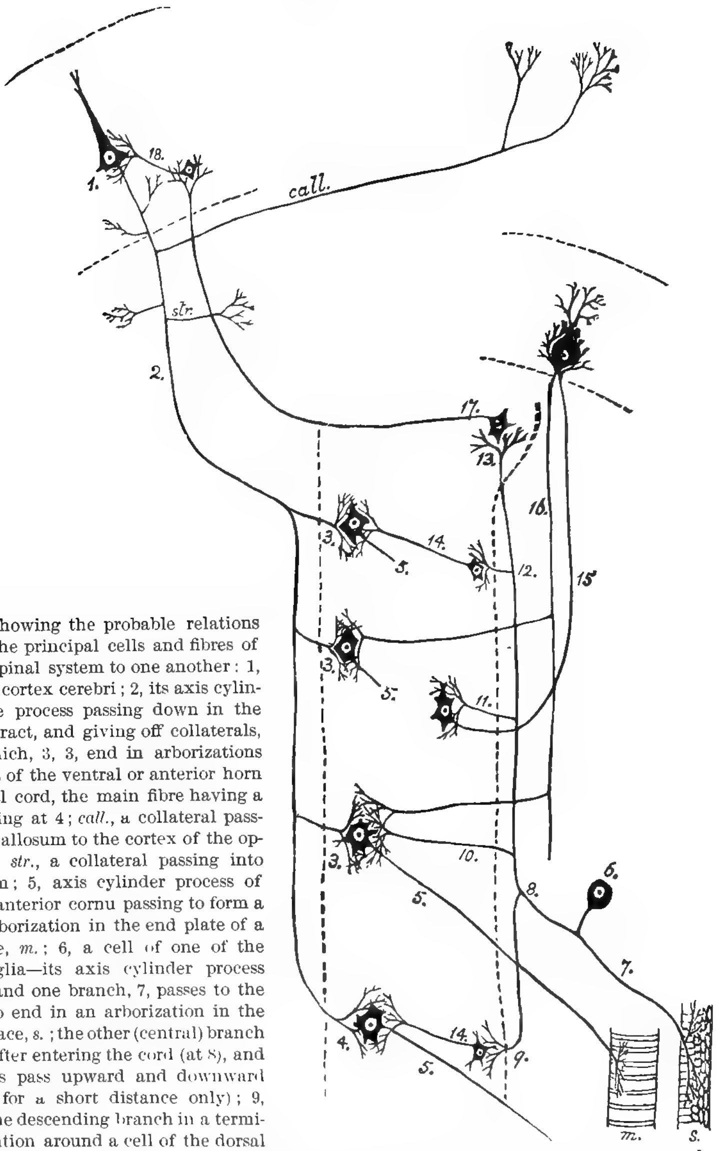


Diagram showing the probable relations of some of the principal cells and fibres of the cerebrospinal system to one another: 1, a cell of the cortex cerebri; 2, its axis cylinder or nerve process passing down in the pyramidal tract, and giving off collaterals, some of which, 3, 3, end in arborizations around cells of the ventral or anterior horn of the spinal cord, the main fibre having a similar ending at 4; *call.*, a collateral passing in the callosum to the cortex of the opposite side; *str.*, a collateral passing into the striatum; 5, axis cylinder process of ventral or anterior cornu passing to form a terminal arborization in the end plate of a muscle fibre, *m.*; 6, a cell of one of the spinal ganglia—its axis cylinder process bifurcates, and one branch, 7, passes to the periphery to end in an arborization in the sensory surface, *s.*; the other (central) branch bifurcates after entering the cord (at 8), and its divisions pass upward and downward (the latter for a short distance only); 9, ending of the descending branch in a terminal arborization around a cell of the dorsal or posterior horn, the axis cylinder process of which again ends in a similar arborization around a cell of the ventral horn; 10, a collateral passing from the ascending division directly to a cell of the ventral horn; 11, one passing to envelop a cell of Clarke's column; 12, a collateral having connections like those of 9; 13, ending of the ascending division of the dorsal root fibre around one of the cells of the dorsal or posterior columns of the bulb; 14, 14, axis cylinder processes of cells of the dorsal horn passing to form an arborization around the motor cells; 15, a fibre of the ascending cerebellar tract passing up to form an arborization around a cell of the cerebellum; 16, axis cylinder process of this cell passing down the bulb and cord, and giving off collaterals to envelop the cells of the ventral horn; 17, axis cylinder process of one of the cells of the posterior column of the bulb passing as a fibre of the fillet to the cerebrum, and forming a terminal arborization around one of the smaller cerebral cells; 18, axis cylinder process of this cell, forming an arborization around the pyramidal cell, 1. (Schäfer, in Quain's Anatomy.)

from below upward, first by the addition of the fibres at successive levels and at the decussation of the fillet, and still higher by fibres from the sensory cranial nerves and nerves of special sensation. These cranial sensory and sensorial nerves are the sensory divisions of the pneumogastric, the glossopharyngeal, and the fifth or trigeminal, with also the acoustic, the optic, and the olfactory. As has already been suggested, various well known cranial ganglia correspond morphologically and functionally to the ganglia of the dorsal spinal roots,—the *petrous* and *plexiform* for the pneumogastric, the *petrous* and *jugular* for the glossopharyngeal, the *Gasserian* and probably other ganglia of the trigeminal distribution for the trigeminal nerve. These ganglia of the cranial nerves have cells which have fundamentally the same structure as those of the spinal ganglia, and all contribute fibres to the great sensory path. They pass from the various cranial ganglia to *terminal sensory nuclei* in the oblongata, and thence, by descending and ascending branches, usually arcuate, into the fillet and other sensory tracts. From the pons to the cortex, the sensory tract in the first part of its course follows a well determined path in the posterior third of the posterior arm of the internal capsule (*carrefour sensitif* of Charcot). Fibres from the sensory tract probably go to the thalamus, which acts as the sensory side of a superior reflex centre; but, whatever may be true with reference to shuntings of the sensory path, the fibres of this tract finally reach the cerebral mantle by way of the tegmental radiations. Here we are concerned only with the consideration of the direct inflows and general course of the tract.

FIG. 111.

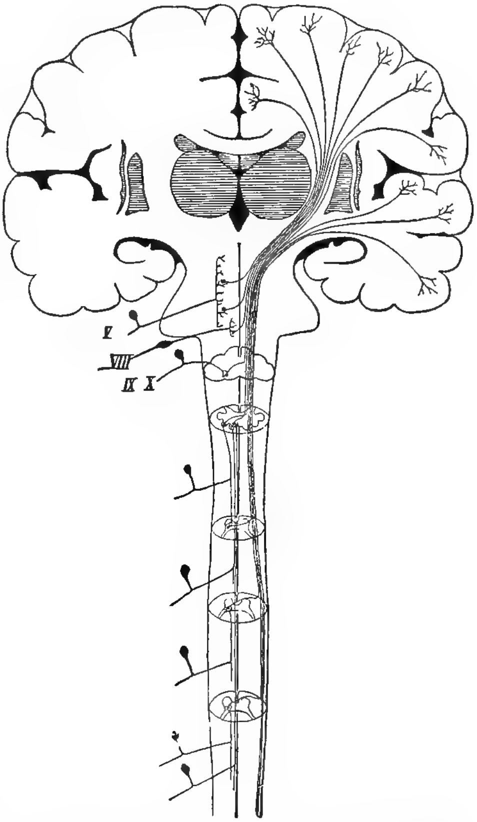


Diagram of the sensory path: *V*, sensory branches of the trigeminal nerve; *VIII*, vestibular branch of the acoustic nerve; *IX*, sensory division of the glossopharyngeal nerve; *X*, sensory division of the pneumogastric nerve. (Van Gehuchten.)

The Cortical Sensory Expanse.—According to the view held by many, the sensory tract terminates in the cortical motor zone, but I believe that the cortical sensory and motor areas are practically separate. After the sensory fibres have passed the internal capsule they begin to spread outward like a great panicle, becoming the *tegmental radiation;* but they still hold their place between the motor region in front and the various more caudal and ventral areas for the special senses. As the tract approaches the cortex, fibres are given off to these surrounding regions; but many sensory fibres reach the cortex of the limbic lobe and the posteroparietal region before their commissural branches pass to other zones. The cortex for common sensation can thus be regarded as a pyramidal mass wedged between other areas and having commissural extensions which put it into relation with the rest of the cortex, the whole forming a far reaching *cortical sensory expanse* which gradually thins out in all directions. *Sensory cells* alone are found in the limbic lobe and posteroparietal region, but their commissural extensions must form various thicknesses and complications of fibres and cells all over the cerebrum. The true *sensory centres* are in the dorsal spinal ganglia and their homologous structures at the base of the brain, the cortical sensory region being simply a terminal expanse. Certain sensory fibres must terminate in the motor cortex, but they do not get there through the pyramidal tract, but by way of commissural cells and fibres issuing from the great sensory tract just before and just after it reaches the cortex. These views tend to harmonize the conflicting opinions of those who regard the cutaneous and allied sensations as represented in distinct cortical regions, and of those who believe in the identity of sensory and motor areas. Lesions affecting that part of the cortex in which sensory cells and fibres alone are present will give the most positive and persistent sensory symptoms, but such symptoms may sometimes result from lesions of the cells and fibres which unite the cerebral portion of the great sensory path with the cortical cells of the pyramidal tract. This question of the cortical representation of common sensibility will be more fully discussed hereafter.

The Paths of the Special Senses.—In order to complete the survey of the sensory side of the mechanism of the nervous system, it is necessary to consider the acoustic, optic, and olfactory pathways,—to compare them with each other and with the paths for the various forms of common sensibility. This can be done by a study of Van Gehuchten's very instructive table showing the constitution of the various sensory tracts and their homologous portions, which I have somewhat modified. The tracts for the special senses, according to the views there summarized, are homologous throughout with the sensory spinal paths of the cranial and mixed nerves; and while future investigations may lead to modifications of some of the views presented by the table, in the main its teachings will stand.

Comparative Table showing the Constitution of the Different Sensory Tracts and their Homologues. (Modified from Van Gehuchten.)

	OPTIC PATH.	ACOUSTIC PATH.	OLFACTORY PATH.	SPINAL SENSORY PATH.	CEREBRAL SENSORY PATH.
PERIPHERAL SENSORY PATH.	Visual cells of retina.	Epithelial (ciliated) cells of the organ of Corti.		Epithelial cells of skin and mucous membrane.	Epithelial cells of skin and mucous membrane.
	Bipolar cells of retina.	Bipolar cells of spiral ganglion.	Bipolar cells of olfactory mucous membrane.	Cells of spinal ganglia.[3]	Cells of ganglia of cranial nerves.[2]
CENTRAL SENSORY PATH.	Ganglionic cells of deep layer of retina.	Multipolar cells of lateral tubercle and of accessory nucleus.	Mitral cells of middle layer of olfactory bulb.	Cells of gray substance of spinal column.	Cells of terminal sensory nuclei of cranial nerves.
	Optic nerves with chiasm.	Trapezium (trapezoid body) and medullary striæ.	Precommissure of forebrain.	Precommissure (anterior commissure) of spinal cord and decussation of fillet in oblongata.	Part of internal arcuate fibres.
	Optic tracts: These divide into	Lateral layers of fillet: These divide into	Olfactory roots and fornix:[1] These divide into	Columns of Burdach and Goll, and Gowers's anterolateral columns in spinal cord; sensory tracts in oblongata: These divide into	Interolivary layer and sensory tracts in pons: These divide into
	Short Paths. (central reflex optic path) to the Pregeminum. Pregeniculum. Pulvinar.(?)	*Short Paths.* (central reflex acoustic path) to the Superior olives, trapezoid nuclei, lateral nucleus, postgeminum, postgeniculum.	*Short Paths.* (reflex central olfactory tract) to the Albicantia and anterior thalamic nucleus.	*Short Paths.* (reflex central spinal sensory tract) to the Different segments of spinal cord.	*Short Paths.* (reflex central cerebral (basal) sensory tract) to the Different gray masses of oblongata, pons, and basal ganglia.(?)
	Long Paths. (central cortical optic path) to the Occipital cortex.	*Long Paths.* (central cortical acoustic path) to the Temporal cortex.	*Long Paths.* (cortical central olfactory tract) to the Uncinate gyre (including amygdala), hippocampal (?) gyre, and dentate (?) fascia.[2]	*Long Paths.* (cortical central spinal tract) to the Thalamus, posteroparietal region, quadrate lobule and gyrus fornicatus.[2]	*Long Paths.* (cortical central cerebral tract) to the Thalamus, posteroparietal region, quadrate lobule and gyrus fornicatus.[2]

[1] The theory advanced by Hill that the fornix is the continuation of the olfactory tract is probably correct. His grounds for this view are given in his translation of Obersteiner's *Anatomy of the Central Nervous Organs*, in a foot-note on pages 345 and 346, and will be referred to later when discussing the functions of the fornix.

[2] Van Gehuchten in his table gives as the cerebral termination of the great sensory path "the cortical motor zone and the cortex of the parietal, occipital, and temporal lobes;" but, according to the views given in this chapter, a sensory region, consisting chiefly of the gyrus fornicatus, quadrate lobule, and posteroparietal gyres, is wedged in between the other cerebral zones. According to the most recent observations, a large portion of the central sensory tract goes to the thalamus on its way to the cortex.

[3] The cells of the spinal ganglia, and of their homologues, the ganglia of the sensory cranial nerves, are morphologically unipolar, but, as has been explained elsewhere, they are embryologically and physiologically bipolar.

The Pyramidal Tract.—The pyramidal tract, Fig. 112, begins in the motor region of the cerebral cortex, which occupies a zone situated on both sides of the central fissure on the lateral and connected mesal aspect of each hemicerebrum. Axis cylinder prolongations of large pyramidal cells of this cortical region descend in the white substance of the hemisphere in this pyramidal tract, and become crowded together in the anterior two thirds of the posterior limb of the internal capsule. Escaping from the internal capsule, the pyramidal tract in the midbrain traverses the white substance of the crusta of the crus in about its middle third, diminishing somewhat in size in its passage, because of the fibres given off to decussate in the raphe to go to the cell nests of the third and fourth nerves on the opposite side of the neuraxis.

FIG. 112.

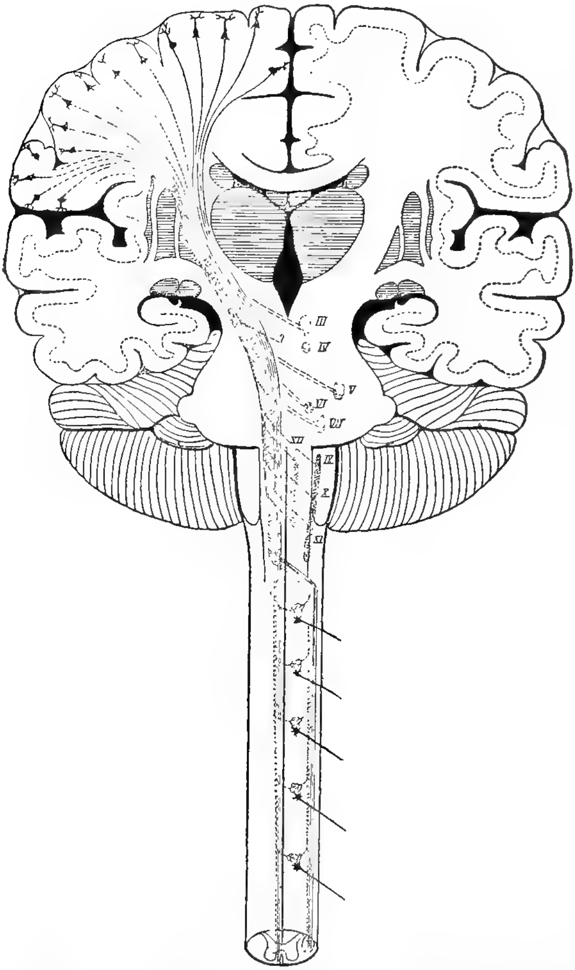
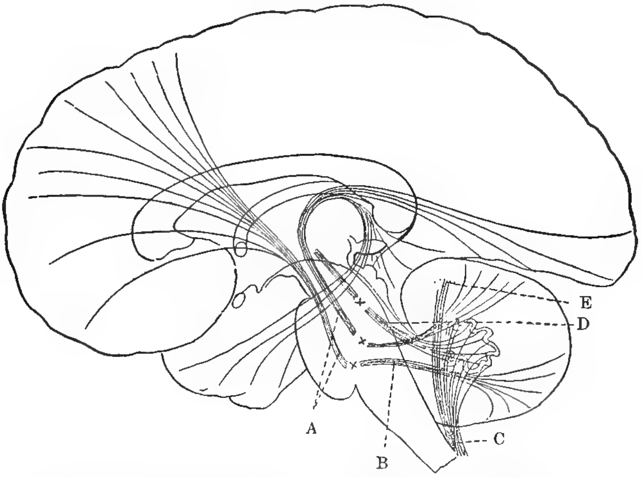


Diagram of the pyramidal path: *III*, common oculomotor nerve; *IV*, pathetic nerve; *V*, motor division of the trigeminal nerve; *VI*, abducens nerve; *IX* and *X*, motor divisions of the glossopharyngeal and pneumogastric nerves; *XI*, spinal accessory nerve; *XII*, hypoglossal nerve. (Van Gehuchten.)

In the cephalic portion of the pons the pyramidal fibres are somewhat commingled with other fasciculi; but near its caudal portion the tract is distinctly defined. It diminishes greatly in its pontile course, fibres leaving it to pass across the median line to the nidi of the motor division of the trigeminal, and of the abducens and facial nerves. In the oblongata it forms the thick bundle of fibres demarcated on the surface of the oblongata as the *anterior pyramid*,

the tract deriving its name from these structures. (Van Gehuchten.) It continues to diminish in size because of the subtraction of fibres which cross the raphe to go to the nucleus ambiguus—the motor nucleus for the glossopharyngeal, pneumogastric, and bulbar portion of the spinal accessory—and to the large nucleus of the hypoglossal. Just at the transition between the oblongata and the spinal cord the pyramidal tracts of the two sides decussate, most of the fibres of each tract crossing to the opposite side and becoming in the spinal cord the *crossed pyramidal tract;* but some fibres pass straight down the anterior column as the *direct pyramidal tract* or *column of Türck.* The former pursues its course to the lower lumbar region, the latter only to the lower portion of the thoracic cord, and each, step by step, diminishes in bulk as, at each segmental level, nerve fibres pass to the anterior horns. The fibres of the direct pyramidal tract probably decussate at successive levels by way of the anterior commissure. An analogy is thus seen between these methods of decussation of the centrifugal motor tracts and the two methods of sensory decussation,—(1) of the tactile fibres in block or mass above the nuclei of Goll and of Burdach, and (2) of the fibres for pain and temperature which ascend possibly in the column of Gowers, after successive spinal crossings; and it thus comes to pass that each half of the highest levels of the nervous system is related for both motion and sensation to the opposite half of the body.

FIG. 113.

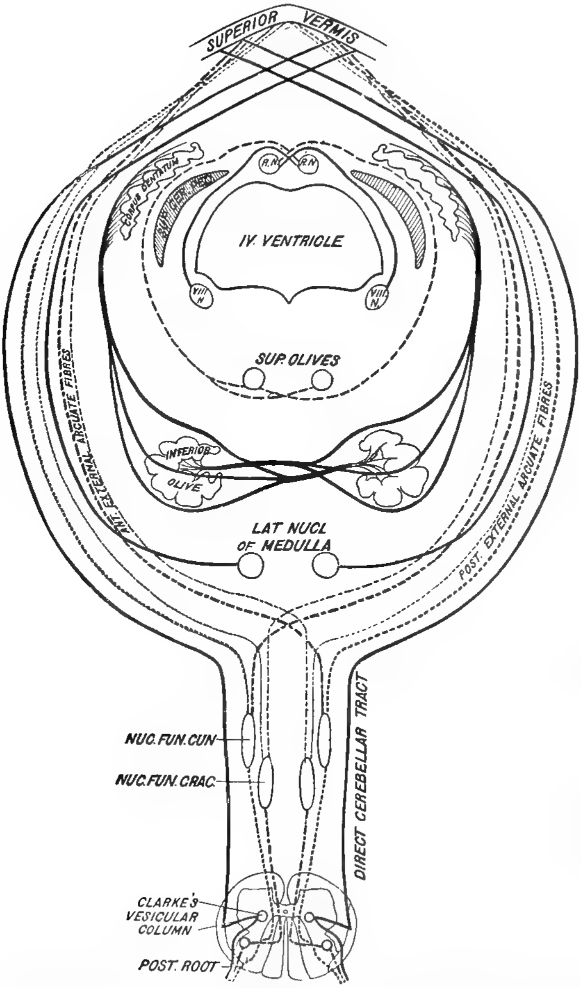


A, anterior and posterior cortico-protuberantial bundles (from the cerebral cortex through the pons to the cerebellar cortex); B, medipeduncle; C, postpeduncle (including bundles from the cerebellum to the oblongata and spinal cord); D, prepeduncle (carrying bundle from dentate nucleus of cerebellum to red nucleus and thalamus); E, roof nucleus. (Van Gehuchten.)

Connections of the Cerebellum.—A study of the diagram (Fig. 113) shows that the cerebellum is connected with the prefrontal cere-

bral cortex by a bundle called the *anterior cortical bundle* or *cortico-protuberantial fasciculus*, which passes through the anterior portion of the anterior arm of the internal capsule to the pons. The cerebellum is also associated with the posterior regions of the cerebrum by a *temporopontile bundle* or *posterior cortico-protuberantial fasciculus*, which passes to the pons from the second and third temporal gyres of the cerebrum through the posterior portion of the posterior limb of the internal capsule. One of these bundles lies ventrad and the other dorsad of the pyramidal tract in the pons. Probably fibres of these tracts terminate in the pontile nuclei, from which others arise, and, after decussating, pass by way of the medipeduncles to the cerebellar cortex. Through the prepeduncle, the dentatum of one hemisphere is united to the red nucleus and perhaps to the thalamus of the opposite side. Cells of the red nucleus have disappeared entirely after transection of this peduncle.

Fig. 114.

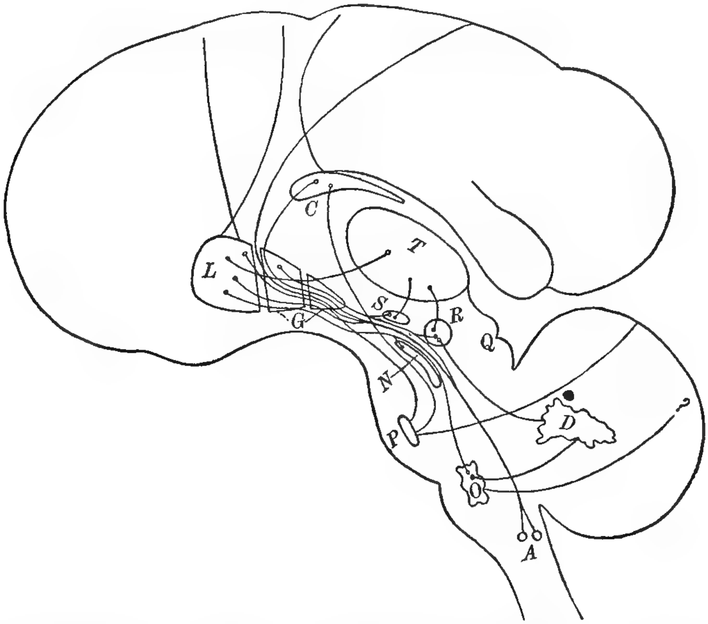


Origins and terminations of the various constituents of the restiform body (postpeduncle). (Bruce.)

By way of the postpeduncle (restis or restiform body), at least two compact bundles go to and from the cerebellum, uniting the latter to the spinal cord and the oblongata. Bruce's diagram, Fig. 114, shows the important tracts which pass through this peduncle. The tracts which occupy its external portion are (1) the

direct cerebellar tract coursing around the outer side of the dentate nucleus and ending in the opposite side of the prevermis (superior vermis); (2) posterior external arcuate fibres, first described by Edinger, which pass from the gracile nucleus and the external cuneate nucleus, and probably end in the prevermis of the side opposite their origin; and (3) a few fibres from the lateral nuclei of the oblongata, which go backward and probably decussate in the vermis. Fibres from the inferior olive pursue a complicated course to terminate chiefly in the dentate nucleus, although some may reach the cortex of the hemispheres. The internal division of the restis consists of two portions, one of which as the external vestibular root of the auditory nerve passes backward along the side of the fourth ventricle to the opposite roof nucleus, the other going outward from the superior olive, also to reach eventually the roof nucleus.

FIG. 115.



Connections of the striata with other parts of the neuraxis: *C*, caudatum; *L*, putamen of lenticula; *G*, pallidum (globus pallidus); *T*, thalamus; *S*, subthalamus; *R*, red nucleus; *N*, intercalatum (substantia nigra); *Q*, quadrigeminum; *O*, inferior olive; *P*, pontile nuclei; *D*, dentatum; *A*, cuneate and clavate nuclei.

Connections of the Striata.—The connections of the striata are as yet imperfectly determined. Edinger, chiefly as the result of studies in comparative anatomy and upon fetal brains, concludes that these bodies or some of their subdivisions are joined to the thalamus, and that bundles of fibres can be traced from them to the oblongata; also that in them certain fibres originate. Other researches and observations have determined a few additional connec-

tions and relations. Comparatively few fibres pass between the striate bodies and the cortex, as would be expected when it is remembered that embryologically both caudatum and lenticula are parts of the cortex. Fibres which originate in the caudatum and the putamen of the lenticula pass through the two inner divisions of the latter to emerge at its base and apex; while others which probably form a portion of the tegmental radiation emerge from the internal capsule and also traverse the globus pallidus, uniting into a bundle below called the *ansa* (*ansa lenticularis*, or *lenticular loop*), which

FIG. 116.

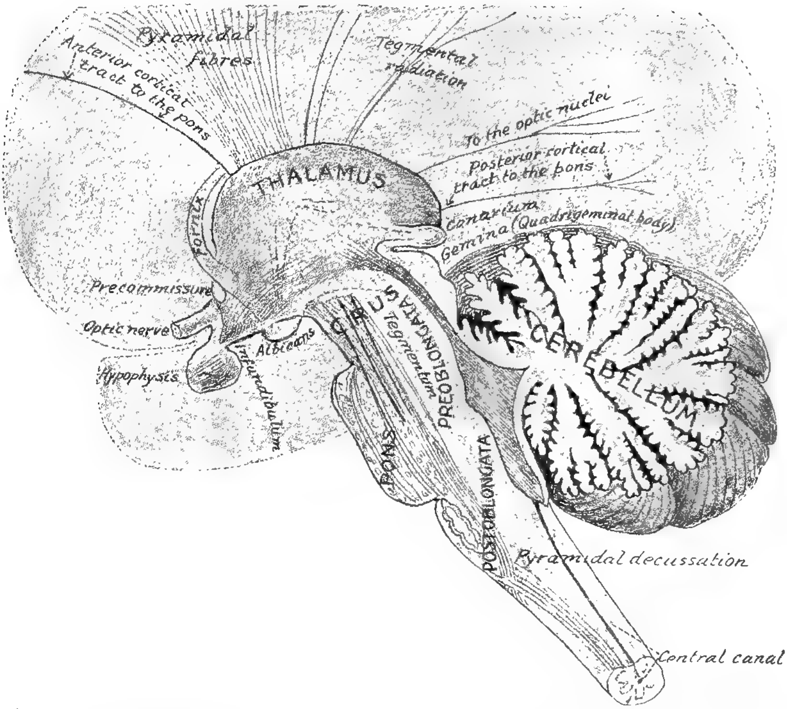


Diagram of a sagittal section through the thalamus and other encephalic structures, showing some of the connections of the thalamus with other parts, and the course of many fibres of the corona radiata. (After Edinger.)

passes into the subthalamic region, in which portions of the striata are connected with the subthalamus, red nucleus, and substantia nigra. The striata also have connections with the pontile nuclei, and with one or both olives, probably with the latter by the central tegmental tract. The numerous and important relations of the striata with the cerebellum are by the way of the red nucleus, the pontile nuclei, the olives, and other basal ganglionic deposits.

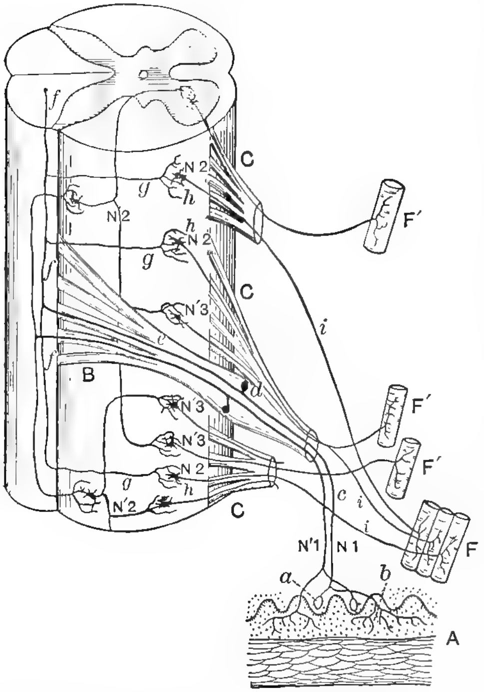
Connections of the Thalamus.—The thalamus has extensive connections, direct or indirect, with almost every part of the neuraxis, chiefly with ascending or sensory tracts; proportionate to its

enormous mass it sends but few fibres downward. (Edinger.) Extensive regions of the thalamus receive fibres from the great ascending sensory tract. Fibres pass between the thalamus and almost all regions of the cortex; and, as stated in describing the connections of the striata, it has intimate relations with these bodies. Numerous fibres pass into it from the internal capsule; and it is related with the tracts for the great nerves of the special senses, through the quadrigeminal and geniculate bodies and other nuclei. The acoustic tract passing through the postgeminum has as the terminus of its short path the postgeniculum. The stratum zonale which spreads over the surface of the thalamus is an expansion of the optic tract, which, passing through the postgeminum, has the pregeniculum for one of its termini. The olfactory tract is anatomically related to the thalamus by the bundle of Vicq d'Azyr, which passes from the albicantia to the anterior thalamic nucleus. The *fasciculus retroflexus* or *retroflected bundle of Meynert* arises from the habenular nucleus and passes backward between the red nuclei to the small *interpeduncular ganglion* between the crura. The thalamus is associated with the clavate and cuneate nuclei, with the olivary body, with the cerebellum, and with other structures. Fig. 116, a sagittal section through the interbrain and other structures, shows some of the important relations of the thalamus, and also indicates the course of some important fibres which do not enter this body. In other diagrams and schemes many important relations of the thalamus are indicated.

Reflex Arcs.—A knowledge of reflex action is fundamental to an understanding of nervous phenomena. Reflex action is effected by means of an afferent or sensory nerve which conveys a stimulus received by the nerve ending to a centre or system of united centres, to be thence transmitted by means of an efferent or motor nerve to the muscles which respond by movement. Fig. 117 is a scheme of such reflex arcs, in accordance with recent views as to the method of transmission of nervous impulses. In the simplest form of reflex action an impulse received by the afferent or sensory side of a spinal centre is carried at once to the nerve cells of the efferent or motor side, and then outward, by means of processes belonging to the latter, to the motorial end plate. Reflex arcs are usually composed only of two elements, and these are found at all levels of the neuraxis from the lowest segment of the sacral cord to the midbrain,—wherever a peripheral sensory terminal can reach a motor root cell. Root fibres from the dorsal ganglia enter the spinal cord, bifurcate in the dorsal or posterior columns, and send out collaterals whose terminals eventually come into contact with motor root cells. Similar simple reflex arcs are constituted through the sensory and motor root fibres of the pneumogastric, glossopharyngeal, and trigeminal nerves.

Compound Reflex Arcs.—Complex or compound reflex paths are formed by a peripheral sensory nerve cell and its central element connecting with several peripheral motor root cells. As, stage by stage, the nervous system is ascended, more and more complex reflex arcs are developed. A great reflex arc is completed through the cerebellum, as shown by studying together Figs. 113, 118, and 119. The sensory central reflex path is by the tracts which pass upward from the nuclei of the columns of Goll and Burdach; while the central reflex motor path is by axis cylinder prolongations of the cerebellar tract which pass by the postpeduncle and antero-lateral columns of the spinal cord to come into relation by their collaterals and terminals with the root cells of the ventral horns. It is supposed that coordination of movements by the cerebellum is particularly brought about by these great reflex paths; and among the most remarkable of complex arcs are those of the reflex optic and acoustic paths described by Held and Van Gehuchten. Even through the thalamus and striate bodies, and by way of the sensory and motor areas of the cortex, higher and still higher reflex arcs are formed. A careful study of these simple, compound, and highly evolved reflex arcs will eventually place on a more substantial basis the doctrine of reflexes as applied to the explanation of nervous disorders; and the first essential to a knowledge of such reflex arcs is an under-

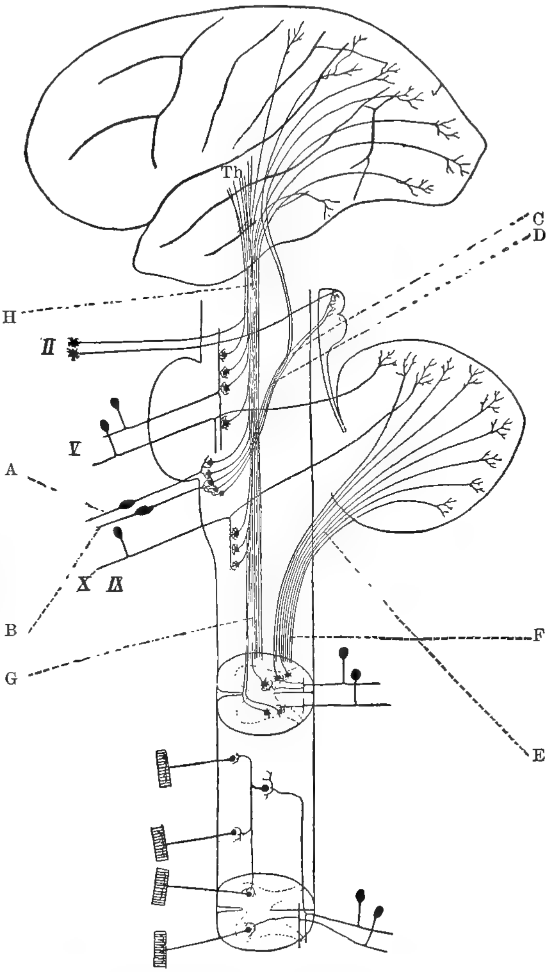
Fig. 117.



Reflex paths of spinal cord: A, skin; B, posterior nerve root; C, C, C, anterior nerve roots; F, F′, muscle fibres. The simple reflex path is composed of N 1, that arises from the periphery by terminal filaments either in an end bulb, *a*, or free between the cells, *b*, and proceeds by a cellulipetal fibre, *c*, to the æsthesioblast, *d*, in the ganglion of the posterior root of a spinal nerve, thence by a cellulifugal process, *e*, which bifurcates, *f*, *f*, in the posterior columns of the cord, sending collaterals, *g*, *g*, *g*, that break into terminals about cells of the anterior horn belonging to nerve cells, N 2. These are composed of short cellulipetal processes that collect impulses; of cell bodies, *h*, *h*, *h*, from which spring cellulifugal axis cylinder processes, *i*, *i*, *i*, that pass to the peripheral muscles through the anterior roots and break into terminal filaments in the motorial end plate. The compound reflex path is composed of a similar collecting nerve cell, N′ 1, one or more correlating nerve cells, N′ 2, and distributing nerve cells, N′ 3. (After Baker.)

standing of their anatomical structure. A study of the table on page 105 which shows the constitution of the different sensory tracts and their homologues will make clear much that relates to these arcs for the nerves of special senses, as well as the other sensory tracts. In Fig. 118 is a scheme of the origin and probable course of the central sensory paths, including the sensory portion of the central reflex arcs through the cerebellum and the quadrigeminal body; and in Fig. 119 is a scheme of the central motor path, including the motor portion of the reflex arcs through various motor cranial nuclei by way of the posterior longitudinal fasciculus, and through the cerebellum by way of the postpeduncle.

FIG. 118.

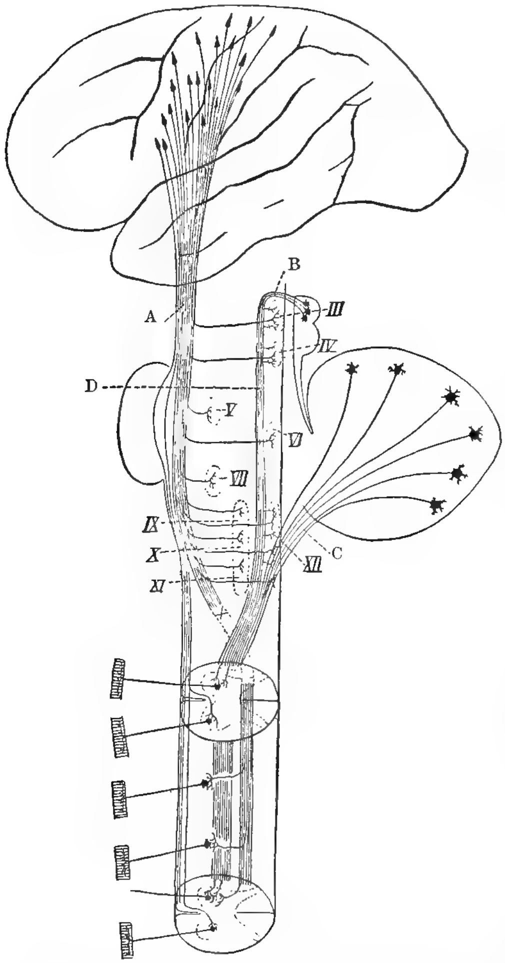


Scheme showing the origin and probable course of the fibres of the central sensory path: II, optic nerve and tract; the Roman numerals V, IX, and X indicate cranial sensory nerves; A, vestibular branch of the acoustic nerve; B, cochlear branch of the acoustic nerve; C, central sensory reflex path of cranial nerves; D, lateral fillet; E, central (cerebellar) sensory reflex path; F, ascending cerebellar tract; G, Gowers's ascending anterolateral tract; H, thalamic and cortical central sensory path; Th, thalamus. (Modified from Van Gehuchten.)

The Projection System. — Meynert regarded the ultimate object of all nervous action to be the projection upon the brain cortex of the images derived from the outside world. The impressions of the body, according to this view, are conveyed to the brain by the ramifications of all the nerves and their terminal organs, and therefore the cerebral cortex is the surface upon which the entire body is projected. This projection system has been

subdivided into an inner, a middle, and an outer portion, the inner connecting the brain cortex with the basal ganglia and neighboring masses, the middle uniting the basal ganglia with the gray matter of the bulb and the spinal cord, and the outer linking the cells of the latter with the periphery of the body. Projection fibres found chiefly in the white matter of the brain and spinal cord are concerned with the conveyance of impulses from the outside world to the sensorium or receptive centres of the brain, or with the transmission of impulses from brain centres to the outside world. They are either *cortico-afferent* fibres, carrying inward and upward impressions from lower levels to the cortex, or *cortico-efferent* fibres, which carry impulses outward and downward from the cortex to lower levels.

FIG. 119.



Scheme showing the origin and probable course of the fibres of the central motor path: the Roman numerals from III to XII indicate motor cranial nerves: A, cortical central motor path; B, reflex (quadrigeminal) central motor path; C, reflex (cerebellar) central motor path; D, dorsal or posterior longitudinal bundle. (Van Gehuchten.)

Levels of the Nervous System.—The cell nests, clusters, and masses of the nervous system are arranged in what have been aptly termed levels, each level having some particular duty at some stage in the progressive working of the entire nervous apparatus. Hughlings Jackson has suggested three evolutionary levels of the nervous system, each sensorimotor, and each representing impressions and movements of all parts of the body. The lowest of these levels is the spinal system of Marshall Hall, including the cord, oblongata, and pons, or rather certain centres in these parts; and from it pass all the nerves of the body. The middle or second

level in its motor province is located in the motor region of the cerebral cortex, and possibly in the striata. The highest or third level is situated in the prefrontal lobe, which Jackson regards as containing centres of representation of the most complex movements of all parts of the body.

Functions of Different Levels.—The cortex of the brain is the seat of the representation of numerous manifestations associated with consciousness ; its different portions subserve highly specialized functions ; in it are stored the memories of past events ; and in it occur the last and highest bodily processes concerned with sensations received from without, and with the setting free of impulses which are projected to lower centres and to the outside world. The thalamus and smaller masses of gray matter in the interior of the brain have functions of importance, but they are not so high as those of the cortex ; part of their work at least is to collect, rearrange, and distribute the impulses and impressions which pass from the cortex downward or from the periphery cortexward. Beginning in the gray deposits at the base of the brain and reaching downward through the spinal cord to its tapering conus are successive areas of gray matter close to the median line in the cord, forming a closed tube. The centres or nuclei in the oblongata, pons, and the more cephalic basal deposits have functions of sensation, motion, and nutrition manifested through the cranial nerves. The spinal centres are preeminently concerned with reflex actions. The cerebellum, like the cerebrum, has surface layers of gray matter, and also interior ganglia which doubtless constitute special levels of nervous activity. Cerebellar centres are correlated with cerebral centres, and both with those of the pons, oblongata, and spinal system. According to the generalization of Spencer and Jackson, the cerebellum regulates the muscular contractions necessary to the maintenance of attitudes in space, and the cerebrum those necessary to effect the changes of attitudes made in response to the successive impressions made upon the organism in time. The functions of special organs and parts will be considered when their lesions are discussed.

Centres.—In neurology the word *centre* frequently occurs. By "centre," as commonly used, is meant simply a collection of gray and white matter—of cells and their processes—of greater or less bulk and complexity, an aggregation which represents physiologically some action. Groupings of centres are spoken of as zones, areas, regions, or districts. Destruction of a centre or zone causes loss or impairment of function ; irritation of it may give rise to abnormal functional activity ; and in numerous ways interference with normal processes is brought about by disturbances of these centres or of the tracts which connect them with each other, with different levels of the nervous system, and with the outside world. The use of the term centre in the description of function and of

disease is largely a matter of convenience. The three things necessary to make of a certain spot a centre are—(1) that its abolition causes certain phenomena to disappear; (2) that its irritation—mechanical, chemical, or electrical—causes the phenomena to be present; and (3) that the part of the nervous system exhibiting these peculiarities is circumscribed in extent. (Ott.) A cortical motor centre in some way represents a definite movement apparently performed volitionally; in a cortical visual centre is the representation of some part of the function of seeing. In studying brain centres, we are, in the language of Hughlings Jackson, investigating the anatomical substrata of visual, tactual, motor, and other ideas, the parts in which occur the most special and widely associated nervous processes which represent impressions of sight, of touch, of movement, and other functions. Lower centres differ from higher in accordance with the simplicity or the complexity of the processes which they subserve.

Bilateral Arrangement of Nerve Centres.—In the higher animals each half of the body is represented and regulated by half of the nervous system. Centres—sensory, motor, vasomotor, and trophic—are in close relation with the origin of every spinal nerve. Sensory and motor nerve centres are arranged in pairs up and down the whole length of the spinal cord with great uniformity, although differing in size and development according to their work. They form a complete series, one for each segment of the cord. The vasomotor and visceral centres lie chiefly between the second thoracic and second lumbar segments. The centres on both sides are more or less intimately connected with each other, in accordance with the greater or less necessity for mutual or reciprocal action. In highly organized parts of the spinal cord, as in the cervical enlargement, the upper thoracic region, and the lumbar enlargement, are centres with special functions. A phrenic centre of great importance to respiration is situated in the upper cervical cord; a pupillary centre or region between the fourth cervical and second thoracic segments; a cremasteric centre in the first lumbar segment; a centre for the patella about the third lumbar; and sexual, vesical, and anal centres in sacral segments.

Oblongatal Centres.—The centres in the oblongata are more important to life than those either in the higher regions of the brain or in the spinal cord, and hence they are sometimes spoken of as *vital*. The three most important of these are the *respiratory*, the *cardiac*, and the *monarchical vasomotor centre*. The respiratory and cardiac centres are located in the terminal nuclei of the vagus nerve, as it is by way of this nerve that the lungs, the heart, and also a large extent of the alimentary canal are supplied. Exactly how the cardiac and respiratory centres are separated from each other is not known. The monarchical vasomotor centre, a group of nerve

cells in the same general locality as the respiratory and cardiac centres, is so called because it exercises a controlling influence over the lower spinal centres. Other centres of less and yet of large importance are a *space* centre for receiving impressions from the semicircular canals, and centres for *deglutition* or *swallowing* and for *phonation*. The various sensations and movements of the head and face are also represented in this region of the glossopharyngeal, facial, auditory, oculomotor, and trigeminal nerves. The respiratory, cardiac, and monarchical vasomotor centres are sometimes spoken of as automatic. In a proper sense this automatic action, and even higher activities, are simply more elaborated forms of reflex action. The oblongatal centres are stimulated by the blood passing through them. Even the ganglia above the oblongata contain centres which show apparent automatism, but the nearer the cortex or brain mantle is approached, the more does voluntary or semivoluntary regulation appear to enter into nervous mechanism.

Higher Cerebral Centres.—Certain centres on the surface of the brain, on its motor or emissive side, are sometimes spoken of as volitional or psychomotor. Stimulation of the areas around the fissure of Rolando gives rise to movement, destruction to loss of movement; but these movements may be reflex or associated, the result of stimulation of sensory centres, and their nature has been variously interpreted. Some of the facts which bear out the view that they are volitional are their definite and purposive character; that they can be reproduced in every detail by ordinary volition; that the paralysis which results from their destruction is that of voluntary motion, in many cases a certain amount of automatic or reflex movement being possible in the paralyzed limbs (Ferrier). Even above and beyond these so-called voluntary motor centres are others of re-representation which exercise such functions as inhibition and attention and the controlling of centres at lower levels.

Inhibition.—Inhibition, by which is meant the checking, stopping, or restraining of function, plays a part of vast importance in the nervous system in health and in disease. If the pneumogastric nerve is cut and a weak electrical current is applied to the cut end farthest from its centre, the beat of the heart will be arrested; in the superior laryngeal branch of this nerve are certain fibres stimulation of which will arrest respiration; and again, if under certain conditions the splanchnic nerve is stimulated, the action of the small intestine will be diminished or stopped. Many reflexes can be suppressed or inhibited through nerve impulses from more or less distant centres or from peripheral parts; but reflex movements which can never be performed voluntarily can never be inhibited. In general terms, most of the phenomena of nervous disease are due either to irritation or destruction of centres or conductors, or to the removal of these from inhibition normally exercised. When restraint is sud-

denly removed, as when a stimulus is suddenly applied or stopped, the resulting overaction or abnormal function is the most intense. The doctrine of inhibition affords an explanation both of some forms of insanity which depend upon disease of the highest regions of the nervous system, and also of some of the simplest forms of nerve disease depending upon changes in nerve endings and nerve fibres. Lauder Brunton believes that inhibition is not dependent upon the existence of special inhibitory centres, but that stimulation and inhibition are different phases of excitement, the two being simply relative conditions depending on the length of the path along which the impulse has to travel and the rate of transmission. The hierarchy of nerve centres, or system of centre-levels, which has been described, is subject to the laws of inhibition. Each set of nerve centres is to a greater or less extent under the inhibitory influence of higher centres; the highest level of the nervous system, supposed to be in the prefrontal region, exercises an inhibitory influence over all the rest, and this can be influenced only by afferent or incoming currents.

Acceleration.—Acceleration of nerve function is under certain circumstances produced by acting upon nerve centres or fibres. It is supposed, for example, that in the oblongata are situated centres which send accelerating fibres to the heart. If the pneumogastric nerves of an animal be divided, stimulation of the oblongata, of the lower end of the divided cervical spinal cord, and of the lower cervical or the upper thoracic ganglion of the sympathetic, will cause acceleration of the heart beat. Exactly what this acceleration means and its relation to inhibitory action are not yet thoroughly understood.

The Dynamics of Nervous Tissues.—Some knowledge of the dynamics of nervous tissues is of fundamental importance to the student of nervous phenomena and nervous disease, who should know something of the stimuli to which nerve cells and fibres react; of the manner and degree of this reaction; of the phenomena by which it is attended; and of the nature of molecular transmission.

The Nature of Nervous Impulses.—According to d'Arsonval, the electrical phenomena of the nerve in action are due to variations in *surface tension* progressing in a wave-like manner along the nerve fibrils; and Schäfer's view that these fibrils may be extremely fine tubules filled with fluid, is in accord with this hypothesis. An understanding of this transmission by variations of surface tension is best obtained by observing what takes place in semifluid living protoplasm. According to Ryder, free and interfacial surface tension is probably the most important factor in determining the shape of cells, and, I might add, in the transmission of energy along the processes and bodies of these cells. What is meant by surface tension is illustrated by reference to a drop of water falling through a

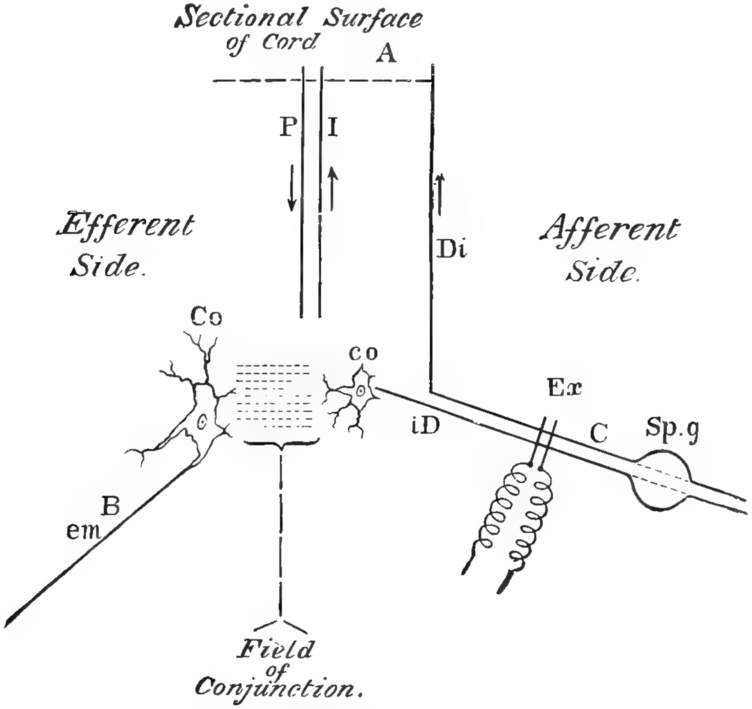
vacuum, which drop will constantly present very nearly the form of a sphere, the molecules of which at every point, being of equal size and having equal powers of attraction, have the same surface tension or reciprocal pull. Any disturbance of this will affect the shape of the drop, but its superficial layers will always form a closed surface. Surface tension is opposed by *viscosity*, or the friction which is developed between moving molecules, and this viscosity differs according to structure. According to the correlation between viscosity and surface tension will be the degree of motion or action exhibited. The motions exhibited by nervous tissues and other forms of living matter are probably of the same character as those shown in the simplest organisms, like the ameba, in which these phenomena are best studied.

Electric Excitation of Nerves.—When a nerve is excited by electricity, chemical, thermal, and other changes take place. The electrical current does not pass up or down the nerve, but across it, and an impulse developed in the nerve is accompanied by its own electrical change. If a nerve is injured, the damaged part becomes electronegative to the rest, so that a current flows from one part to the other. This is called the *negative variation* or action current. This is the current evoked by nerve impulses which pass down a nerve which has been cut, as can be shown when the cut end and the longitudinal surface of the nerve are connected respectively with a galvanometer. Nerve conductivity is increased by sending an electrical current through the nerves. Electrotonus, a state of modified neuro-irritability, is produced, and can be spread by contact from nerve to nerve. Heat appears to improve conductivity, that is, to diminish resistance, and cold to do the opposite, except that, according to Gotch, cold directly applied to the nerve trunk has not this effect. When an electrical current not quite sufficient to excite a muscle to contract is sent along the nerve, if a heated bar be brought near the nerve, the muscle will contract. Nerves are more easily excited by electrical and other stimuli the nearer they are to their centres. The most important of these stimuli, besides the electrical, are mechanical, chemical, and thermal.

Transmission of Nerve Impulses.—The average rate at which nervous impulses are conveyed is about one hundred feet or thirty-three metres per second, varying somewhat for different nerves and under special circumstances. It is of great interest to know whether the same nerve can carry different impressions at the same time, which would be in accordance with the transmission of electricity. One of the most valuable commercial discoveries is that of multiplex telegraphy, by which numerous messages can be sent along the same wire simultaneously. The study of questions such as this may eventually lead to important discoveries regarding the so-called forms of sensation. New light has been thrown on nervous impulses by

the investigations which have developed the theory of nerve trees, and the transmission by contact from nerve cells to nerve cells, which is analogous to methods of electrical transmission. Horsley with others holds that it is probable that all nerve fibres transmit impulses at the same rate, but this view is by no means universally held. It is supposed by some physiologists that afferent or sensory impulses travel at a quicker rate than efferent or motor impulses. Exner has suggested that the rapidity of transmission in cells and

Fig. 120.

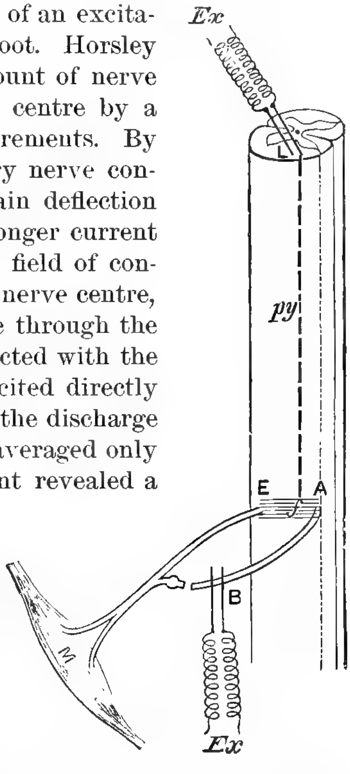


Scheme of a simple nerve centre in the spinal cord: Sp.g, ganglion on the dorsal or posterior root; C, posterior root; Ex, excitation electrodes applied to the posterior root; iD, indirect nerve sensory or afferent path; Di, direct afferent path ascending cord in posterior column; co, small corpuscle of posterior part of gray matter; I, internuncial fibre; P, pyramidal tract fibre from brain; Co, large corpuscle of ventral or anterior horn of gray matter; B, anterior root. (Horsley.)

fibres of nerve centres is different from that in peripheral nerves. Horsley and Gotch, having examined this point experimentally, came to the conclusion, provisionally at least, that the fibres in the spinal cord conduct like those of the peripheral nerves. It is important, as Horsley states, to determine the rate of the transmission of impulses in the peripheral nervous system, as otherwise it would not be possible to measure the amount of time a nerve centre occupies in its work.

The Action of Simple Nerve Centres.—The structure of a simple nerve centre such as is present in the spinal cord may be regarded, as stated by Horsley, as consisting of three parts: (1) an afferent side for the reception of impressions; (2) a field of conjunction, which connects the afferent side with (3) an efferent side, through which impulses pass out down the nerves. Fig. 120 is a diagrammatic representation by Horsley of a simple nerve centre in the spinal cord, and of its relations to the periphery and to the brain; also of an excitation electrode applied to the posterior root. Horsley and Gotch obtained an idea of the amount of nerve energy discharged by a spinal nerve centre by a system of comparative electrical measurements. By exciting directly an afferent or sensory nerve connected with the galvanometer, a certain deflection was obtained; then by means of a stronger current an excitation was conveyed across the field of conjunction through the efferent or motor nerve centre, and this centre was made to discharge through the efferent nerves, which also were connected with the galvanometer. The sensory nerve excited directly gave a deflection of 200 to 300, while the discharge of the nerve centre on the nerve fibre averaged only 26 on the same scale. This experiment revealed a block to the passage of the current from the afferent to the efferent periphery. The actual time taken in converting a sensory into a motor impulse is approximately arrived at by an extension of this electrical method, which is indicated in Fig. 121. A sensory nerve fibre is stimulated at B, and the time between this stimulation and the contraction of the muscle at M is accurately measured. A represents the afferent or sensory side, and E the efferent or motor side, of the simple spinal centre. The time taken to pass from B to A and from E to M is subtracted from the whole time lost in going from A to M, and in addition about one-hundredth of a second as representing the time lost, according to Helmholtz, in the motor nerve ending before the muscle contracts. Horsley and Gotch found as the result of their experiments that the time lost in the nerve centres averaged .006 of a second; but other observers have found it much longer, Exner making it as much as the six-hundredth of a second: that a definite time is lost is conceded by all.

Fig. 121.



Experiment to show the time taken by a nerve cell to convert a sensory into a motor impulse. (Horsley.)

CHEMISTRY OF THE NERVOUS SYSTEM.

Chemical Composition of Nervous Tissues.—It is commonly assumed that the composition of the tissues of the nervous system, especially of the gray matter of the brain, is the most complex within the domain of organic chemistry. The numerous functions of the brain, and the great rapidity with which these are exercised, together with the profound mystery that still surrounds the phenomena of mental action, lead to the supposition of high complexity of chemical structure; but it is probable that the chemical character of the tissues of the ovum is equally complicated, and that the processes of transformation involving the action of enzyms, or formless ferments, are as intricate as any in brain structure. While considerable work has been done in the way of extracting definite crystallizable principles from brain structure, we have as yet no positive evidence that such principles are concerned in brain functions. They may be merely products of the breakdown of the proteid structures, and therefore may be of no more significance than the urea and uric acid found in the examination of the tissues of a kidney.

Chemical Characteristics of Nervous Tissues.—Nervous tissues in a negative or passive state are feebly alkaline; when in a condition of activity they become acid in reaction; and after death also the nervous substance usually rapidly becomes acid. The fresh gray matter of the brain is alkaline. The percentage of water in the nervous system varies according to the portion of the system, whether nerve cells or nerve fibres. The gray substance of the human brain contains about eighty-two per cent. of water, expelled at 95° C.; the white matter, about sixty-eight per cent. The nerves contain less water than the centres, spinal and cerebral, averaging between sixty-five and seventy per cent. The following table, from Landois and Stirling's Physiology, shows the approximate composition of the two classes of nervous tissue:

Chemical Composition.	Gray Matter.	White Matter.
Water	81.6 per cent.	68.4 per cent.
Solids	18.4 "	31.6 "
	100.0	100.0
The solids consist of:		
Proteids	55.4 "	24.7 "
Lecithin	17.2 "	9.9 "
Cholesterin and fats	18.7 "	52.1 "
Cerebrin	0.5 "	9.5 "
Substances insoluble in ether	6.7 "	3.3 "
Salts	1.5 "	0.5 "
	100.0	100.0

Chemical Constitution of the Brain.—We owe a large part of our knowledge of this topic to Thudichum, according to whom the brain as a whole is an aggregated mass of bioplasm, which derives its peculiarity mainly from specific chemical additions. The stroma of bioplasm is mainly constructed of proteid substances, in which the specific matters are distributed or with which they are combined in such a manner as to produce the living brain tissue or *neuroplasm*. It contains small quantities of *soluble albumin*, also *fibrin*, and a substance termed *neuroplastin*. The nuclei of the ganglionic cells are composed of a substance which Thudichum calls *gangliocytin*, which, as it contains phosphorus, is also termed *cytophosphatide;* its congeners in the yeast cells bear the name *nuclein*. The relative weight of the proteid matters of the brain free from its membranes amounts to seven to eight per cent., varying in different parts of the brain.

Chemical Constituents of the Brain.—The immediate principles, organic and inorganic, which have been isolated from the brain may be arranged into (1) nitrogenous phosphorized principles; (2) nitrogenous non-phosphorized principles; (3) true proteids or substances closely allied thereto; (4) principles containing carbon, hydrogen, and oxygen, which include alcohols, carbohydrates, and organic acids; (5) inorganic bodies, which include acids, bases, and salts, which may either be free or in combination with the foregoing organic principles. Much study has been spent on the phosphatides or phosphorized bodies of the brain; they are analogous to the phosphates, and may be regarded as complex derivatives of phosphoric acid. One class of phosphorized principles, the *lecithins*, is said to be present in every human brain to the extent of at least sixteen grams. Numerous other principles have been isolated, as have also nitrogenized and non-phosphorized substances. *Cholesterol*, an important constituent of gall stones, in quantity and possibly in function, is also an important constituent of the brain; it is an alcohol, solid at ordinary temperatures and possessing a distinct crystalline form. Lactic acid is the principal organic acid of the brain.

Typical Brain Principles.—It is impossible to present clearly and fully the chemical composition of brain principles without formulas, but it will suffice to indicate the nature of a few of the important types of compounds. The lecithins contain the radical phosphoric acid associated with an ammonium derivative partly alcoholic in structure, and also with an incomplete fat. Different lecithins may be produced by radicals of different acids. It is possible that the character of the fatty food entering into the diet may exercise a direct influence on the composition of the lecithin, and thus indirectly upon mental action. The *kephalins* are analogous to the lecithins in composition, but contain a radical of a peculiar acid,

kephalic acid. The principal form, which is present in very large quantity, is the most unstable of all the phosphorized brain principles. The substances consisting of carbon, hydrogen, and oxygen are doubtless the results of the breaking down of fundamental structures, and are therefore excretory products. Among the nitrogenous principles are *phrenosin* and *cerebrin*, from which by the action of dilute acid a true sugar, *galactose*, is obtained.

Pathological Changes in the Brain viewed Chemically.—Glycerophosphoric, oleic, and other fatty acids have been extracted from the softened portions of the brain. In disease, not only do normal constituents and ingredients decompose, but abnormal products accumulate through local action or by way of the blood. In this way poisons such as arsenic, antimony, and narcotics affect the nerve centres. In cholera, Thudichum found in the cerebrospinal fluid two per cent. of urea. Blyth has shown that in chronic lead poisoning there is often a substitution of one or more lead atoms for the hydrogen of the kephalin, and it is obvious that the functions of such a molecule will be materially altered by this change in composition. As phosphorus and nitrogen, prominent ingredients in the brain principles, are analogous in their chemical relationships to arsenic and antimony, the substitution of the latter for the phosphorus and nitrogen of the brain principles might occur in poisoning by their compounds. It has been asserted that the injection of arsenic in large doses produces increased excretion of phosphates, indicating a direct substitution of one element for the other, but the experimental evidence for this view is not entirely satisfactory. That excessive excretion of phosphates in urine is dependent on brain disease is not established.

Nervous Action and Chemical Processes.—The actions which take place in nerve cells are known to be accompanied by chemical processes. If the gray matter of nerve centres is stimulated by drugs or by other methods, it becomes acid in reaction. Mosso has shown that activity in nerve cells is accompanied by a rise in temperature. Whether nerve fibres take part in the chemical and thermal changes of nerve cells has been doubted. Fatigue is believed to be due to the accumulation of the products of chemical action: it is held that the results of this action are felt only by the gray matter, because it is thought that nerve fibres cannot be fatigued like nerve cells. Edes, however, has shown that the action current which can be studied in nerves after some hours of activity tends to become weakened. Rolleston was unable to obtain any evidence of the development of heat by the passage of the nerve impulses along nerve fibres; but, as suggested by Schäfer, the thermal changes in the excited living nerve may have been too slight to be detected. The view of Schäfer, that the same chemical processes go on in the axis cylinders as in the gray substance, is probably correct.

CHAPTER II.

GENERAL PATHOLOGY AND ETIOLOGY, SYMPTOMATOLOGY AND METHODS OF INVESTIGATION, ELECTRICITY, AND GENERAL THERAPEUTICS.

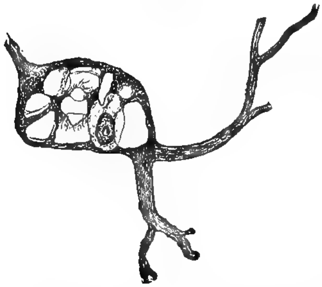
GENERAL PATHOLOGY AND ETIOLOGY.

Pathological Classification of Nervous Diseases.—Diseases of the nervous system can be included under a few general heads, as (1) developmental malformations; (2) inflammations; (3) degenerations; (4) tumors and adventitious products; (5) diseases of blood-vessels; (6) vascular disturbances; and (7) functional disorders. Lesions of the nervous system may be macroscopic, microscopic, or invisible to all known methods of investigation. Under coarse macroscopic disease are embraced inflammation, hemorrhage, softening, abscess, tumors, parasites, and fibrosis. Some of the lesions ordinarily regarded as microscopic, as the scleroses, are determinable by the naked eye, but need microscopic investigation for their full comprehension. Some affections long regarded as undemonstrable have yielded to new methods of research. A helpful method of considering nervous pathology is by relating the special phenomena to their localized lesions. Cerebral and spinal disease give symptoms of local irritation, destruction, pressure, and instability.

Developmental Malformations.—The development of the nervous system may be arrested, or it may be aberrant, causing *agenesis* or *aplasia*, and giving rise to malformations and monstrosities. Arrests and deviations of growth and development act upon both the physical structure and the psychical nature; so that mental deficiency usually accompanies both arrest and abnormal increase of development. Forms of idiocy and imbecility, dwarfism, gigantism, acromegaly, cretinism, and similar affections are sufficient evidences of the truth of this statement. Arrests of development usually occur during fetal life, but both arrests and deviations may take place at any period. When not fetal they are most likely to occur before adolescence. The entire cerebrum may be undeveloped; or gyres and lobes may have disappeared, leaving a cavity in the brain; or the brain and spinal cord may be unusually small. Hypertrophy of the brain is a rare condition, and may be total or partial. Normal tissues, sometimes the gray matter and more rarely the white substance, may be misplaced. The cavities of the brain and of the central spinal canal may be dilated. Occasionally the bony encasement of the brain and cord may be undeveloped.

Inflammation.—Inflammation, the response of living tissues to an irritant accompanied by abnormal nutritive processes, may attack any part of the nervous system or its envelopes. Primarily it is an affair of bloodvessels and lymphatics, but the nervous tissues may be affected either rapidly or slowly. Inflammation may be *parenchymatous*, affecting the specific textures of a part, or *interstitial*, involving the tissues which intervene between the true structural elements; or it may be *diffused*, attacking both parenchyma and non-nervous tissues. When neuritis is parenchymatous nerve fibres are involved, when interstitial the connective tissue and nerve sheaths, and when diffused all parts of the nerve bundle. It is sometimes difficult to decide whether well known affections are primarily parenchymatous or interstitial inflammations. How far inflammatory processes play a part in the degenerations of nerves and of the white matter of the spinal cord is a mooted question. Inflammation sometimes leads to degeneration, but it may pass away without this result, and degeneration may arise without preceding inflammation. Vacuolation (Fig. 122) is sometimes a result of inflammation, but may also be due to postmortem changes. Inflammation is commonly of infectious or toxic origin.

Fig. 122.



Cell of anterior horn with ten vacuoles in myelitis. (Obersteiner.)

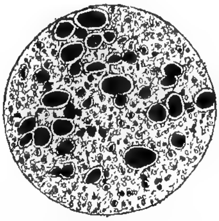
Varieties of Inflammation.—Inflammation of nervous tissues may be *exudative*, and according to the nature of the exudate may be subdivided into serous. fibrinous, or purulent. Any of these may or may not be accompanied by a necrosis or death of the tissues. Inflammatory exudates may infiltrate the substance of the brain, spinal cord, or nerves. Inflammation also may be focal, localized to one spot or region; or it may be multiple or disseminated; or it may be diffused continuously over a large area. A multiple encephalitis is found in some acute insanities, and disseminated myelitis is a not infrequent infectious disorder; in rare cases patches of inflammation may be scattered everywhere throughout the neuraxis. When inflammation attacks the gray matter of the spinal cord or brain, it is usually acute, and is called *poliomyelitis* or *polioencephalitis*. Inflammation dependent upon syphilis or other infectious agencies is described as *proliferative* when new tissue is formed slowly without exudation. Neural inflammation may be acute, subacute, or chronic.

Meningitis.—*Meningitis*, or inflammation of the membranes which envelop the brain and spinal cord, is usually included under affections of the nervous system. The inflammation may be of the dura, when it is termed *pachymeningitis*, or it may be of the pia and

arachnoid, when it is known as *leptomeningitis*. Like inflammation of nervous tissues, it may be exudative or proliferative; focal, disseminated, or diffused; acute, subacute, or chronic. When subjacent nervous texture is involved with the membrane, a *meningo-encephalitis* or *meningomyelitis* results.

Degenerations.—Degeneration of nervous tissue does not occur spontaneously, but originates either from alteration of the bodies of the nerve cells which are trophic centres, or by separation of these centres from their processes. As commonly understood, it is a gradual process of decay and death of the nerve cells and fibres; although the term *acute degeneration* is sometimes applied to rapid processes of necrosis or softening. Degenerations may be *colloid, granular* or *fatty, pigmentary, fibroid,* or *calcareous.* In colloid degeneration minute round or ovoid bodies permeate the nervous textures. Granular or fatty disintegration of nerve cells is observed particularly in the brain cortex; cells become displaced or are represented by collections of granules, much fatty matter being present, and especially around the bloodvessels. In pigmentary degeneration the cells are deeply pigmented, but it must not be forgotten that pigmentation is present in healthy nerve cells. In fibroid degeneration fibrous tissue is substituted for the nerve elements; and in calcareous degeneration the deposition of insoluble compounds of calcium and magnesium takes place.

FIG. 123.

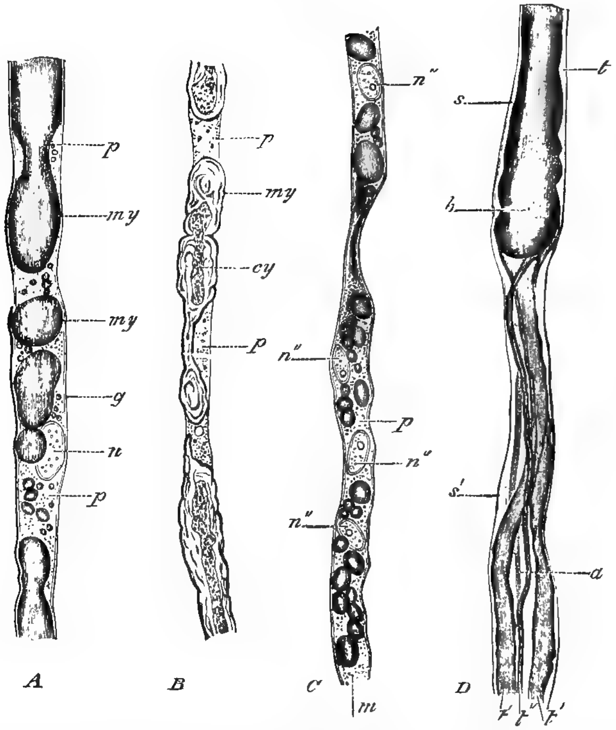


Section from the spinal cord of a case of myelitis. The neuraxons are in the early stages of degeneration and are of various sizes, some being greatly enlarged, others of normal size.

Primary and Secondary Degenerations.—The most important classification of degeneration of nervous textures is into *primary* and *secondary.* Simple atrophy which may attack the gray matter of the cord or brain is a form of primary degeneration. The cell bodies diminish in size and lose their outlines, and their processes dwindle or disappear. Primary degeneration may be due to embryonal arrest, or it may be infectious or toxic. Secondary degeneration is commonly caused by the separation of nerve processes from their cell bodies or trophic centres; but, according to Vulpian, it may be due to persistent irritation of nerve fibres; and it may result also from the starvation of the nervous tissue through the cutting off of blood supply. Primary and secondary degeneration show marked histological differences. The lesion in primary degeneration is usually histochemical. The nerve fibres are not always absolutely disintegrated, and hence recovery is sometimes possible when the toxic influence is removed. (Vassale.) As usually studied, secondary degeneration follows such focal diseases of the brain or cord as hemorrhage, softening, tumor, or abscess; or a spinal injury,

or a compressing curvature. If a function is interfered with by a focal lesion, degeneration takes place in the nerve tracts along which the impulses which interpret this function are conveyed; and secondary degenerations are therefore divided into *descending* and *ascending*, or *centripetal* and *centrifugal*, or, using the terminology of Ramón y Cajal, *cellulipetal* and *cellulifugal*. A destructive lesion of the cortical motor centres causes descending degeneration in the pyramidal tract as far as the ventral horns of the cord. Ascending degenerations are chiefly in the line of the sensory tracts in the spinal cord, but any nerve tract, long or short, may secondarily degenerate.

Fig. 124.



Degeneration and regeneration of nerve fibres in the rabbit: *A*, part of a nerve fibre in which degeneration is commencing in consequence of section (fifty hours previously) of the trunk of the nerve higher up; *my*, medullary sheath becoming broken up into drops of myelin; *p*, granular protoplasmic substance which is replacing the myelin; *n*, nucleus not yet multiplied; *g*, primitive sheath. *B*, another nerve fibre in which degeneration is proceeding, the nerve having been cut four days previously. This specimen is differently prepared from the others, so as to exhibit the axis cylinder (*cy*) also partly broken up into portions of different lengths enclosed in the myelin, *my*. *C*, more advanced stage of degeneration, the medullary sheath having in great measure disappeared, while several nuclei (*n″*, *n″*) have been formed by division of the single nucleus of the internode. *D*, commencing regeneration of a nerve fibre; several small nerve fibres (*t′*, *t″*) have sprouted out from the enlarged cut end (*b*) of the nerve fibre (*t*); *a*, an axis cylinder which has not yet acquired a medullary sheath; *s*, *s′*, primitive sheath. (Quain's Anatomy, after Ranvier.)

Wallerian Degeneration.—Waller showed that division of a spinal nerve was followed by degeneration. When the ventral root

was divided before it joined the dorsal, only the distal motor fibres underwent degeneration; while if the dorsal or posterior root was cut between the ganglion and the spinal cord, only that portion of the nerve root which passed between the point of section and the spinal cord degenerated. These experiments proved that the trophic centres for the sensory nerves were situated in the posterior ganglia, and those for the motor roots in the multipolar nerve cells of the ventral horns of the cord. Sensory peripheral nerves which spring from cells in the posterior spinal ganglia degenerate downward and outward, and conduct upward and inward, an apparent exception to the usual rule, but one explained by the law of Waller. Friedländer, Krause, and Marie record facts regarding degeneration of the nerves and spinal cord following the amputation of a limb which are apparently not in accord with this law. After amputation of the thigh, Marie found that the posterior columns of the cord on the side of the amputation presented the most constant and decided diminution, but the opposite side also showed marked decrease in the posterior spinal columns, and to a less extent of the anterolateral region. He suggested that both ascending Wallerian degeneration and ascending neuritis of septic origin may have occurred, the amputation having taken place before the days of antiseptic surgery.

Regeneration.—Nerve cells sometimes appear to regenerate, but the destruction in these cases has not been complete. Even after a long time regeneration in peripheral nerves may occur, the activity and completeness of the process depending on the influence which can be exerted by their trophic centres. Suturing of nerves has resulted in the restoration of function after months and years. The process is largely one of outbudding and outgrowing of the central portion of the nerve, which is in accordance with the facts of embryology, all nerves being outgrowths from the central nervous system. Nerve tracts in the spinal cord and brain have power to regenerate, but this is not so great as in the peripheral nerves, and yet even old cases of compression of the spinal cord may make great improvement after a long time, largely through the regeneration of the columns of the cord.

Vascular Origin of Spinal Degeneration.—Impoverished blood may cause degenerations of the central nervous system, as is demonstrated by the posterior sclerosis of pernicious anemia. Even momentary arrest of circulation will cause degenerative lesions. When the circulation in the abdominal aorta was temporarily suspended and then reëstablished, Ehrlich and Brieger found considerable degeneration of the gray substance related to the anterolateral columns. The bloodvessels in spinal sclerosis are decidedly more prominent than in secondary systemic degenerations, and the lesions are most decided in the vicinity of these altered vessels. (Marie.) Brazzola, in a case of general paralysis of the spinal type,

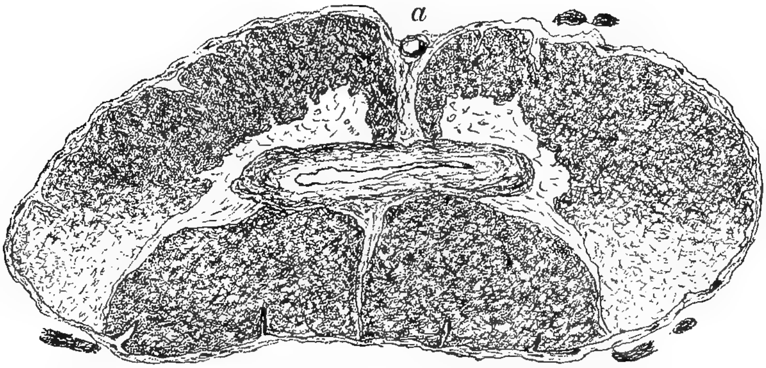
found everywhere decided vascular alterations from which the mischief appeared to take its start. The changes in the nervous elements may in some cases be due directly to the action of a specific virus or toxin, and in others to preceding inflammatory and degenerative alterations in the bloodvessels. According to Preston, in posterior spinal sclerosis a diminished amount of blood is sent to the spinal cord, owing to arterio-sclerosis, and, as a result of the imperfect supply of nutriment, atrophy and degeneration occur.

Sclerosis.—Certain organic nervous diseases are usually classed as *scleroses.* The term sclerosis comes from a Greek word meaning "hard," and in using it cause and effect are often confused. The word is more applicable to the results of the disease process, the fibroid induration, than to the disease itself, although it is used in both senses. Under the scleroses are included such diseases as posterior sclerosis or locomotor ataxia, lateral sclerosis or spastic paraplegia, combined sclerosis or ataxic paraplegia, disseminated and amyotrophic lateral sclerosis. Some regard sclerosis as a chronic inflammatory process affecting the nerve elements, a true parenchymatous inflammation ; others as a disease in which a proliferation of neuroglia is the primary process. Marie has shown that the lesions of the lateral columns in amyotrophic sclerosis are largely due to alterations of cells situated in the central cinerea of the spinal cord ; and he believes that in other forms of sclerosis, in paretic dementia, and in pellagra, the lesions of the white fasciculi of the cord are poliomyelitic in origin. The existence of cells whose prolongations go to both the lateral and the dorsal columns explains the coincidence of lesions in various columns producing the "mixed" or "combined" forms of sclerosis. Tabes is probably primarily an exogenous affection, the cells of the posterior ganglia being first attacked. Recent researches by Popoff on the histology of disseminated sclerosis led him to the conclusion that the sclerotic tissues are products which result from the destruction of nerve fibres, and that no development of connective tissue takes place ; also that the axis cylinders as well as the medullary sheaths are involved, and that the bloodvessels are the starting points.

Gliomatosis.—The process known as gliomatosis or gliosis is of special importance in connection with the disease known as syringomyelia. The development of the neuroglia has already been considered. An undue proliferation of the glia cells of this tissue constitutes gliosis or gliomatosis. The neurogliar lining of the spinal cavity in the embryo may not undergo the usual modification. Gliomatosis, as commonly seen, is practically a neoplastic process, and may be regarded as essentially a chronic inflammation of the gray matter, the cavities and other conditions being secondary. Cavities are not always present in gliomatosis, or the cavity formations may be few and small. In syringomyelia the cavities

are often surrounded by gliomatous tissue which contains glia cells in all stages of development and regeneration, and more or less broken down, as shown in the illustration (Fig. 125).

FIG. 125.



Section from the thoracic region in a case of syringomyelia. The gliomatous tissue is broken down in the centre and has given place to a cavity : *a*, ventral sulcus.

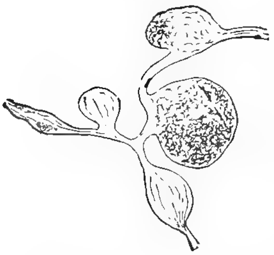
Senility.—In normal old age the nervous system has undergone and is still undergoing a process of slow decay. The brain shrinks, the cortical cinerea becoming thinner and the fissures shallower, and the shrinking is compensated for by an increase of ventricular and subarachnoid fluids. In the spinal cord and in nerve trunks many of the medullary sheaths disappear. Nerve conductivity is lowered. Bloodvessels undergo changes. In some of the degenerative affections of old age the pathological conditions present are simply exaggerations of ordinary senility, but the lesions may be most pronounced in certain portions of the neuraxis. In paralysis agitans the nervous system tends to early degeneration,—to premature senility, in other words.

Tumors and Adventitious Products.—Statistics give somewhat different results as to the relative frequency of different forms of intracranial and intraspinal tumors, but the tuberculous, gliomatous, sarcomatous, and syphilitic are of frequent occurrence. Hydatids and serous and hemorrhagic cysts are comparatively common. True gliomata are simply outgrowths of neuroglia. When carcinoma attacks the nervous system it is almost invariably secondary. Many, if not most, of the neuraxial neoplasms originate in the membranes of the brain and spinal cord. Several varieties of neuromata or tumors connected with the nerves are known.

Vascular Diseases and Disturbances.—Hemorrhages, arterial and capillary, show a tendency to select certain parts of the nervous system, and particularly of the brain. In cases of cephalic hemor-

rhage, enlargements of the arteries called miliary aneurisms are common. Softening is generally due to occlusions of bloodvessels. Vegetations, detritus, or coagula, carried from the heart or large vessels, plug the arteries of the brain and thus lead to softenings; and sometimes around a hemorrhage, tumor, or exudation, local ischemia and softening take place. Softening is also caused by compression or by contusions and concussions of the brain and cord. Either active or passive congestions may occur in the nervous system. The causes of the latter are mainly mechanical. Active congestion may be the result of excitement, fever, or the use of alcohol or other toxic agents. Anemia of the brain or cord may be part of a general condition of anemia, or it may be due to profuse hemorrhage or exhausting disease. Local anemia is sometimes caused by alterations in the calibre of the vessels. *Functional disorders* include a gradually decreasing list of diseases, the pathology of which is not certainly determined.

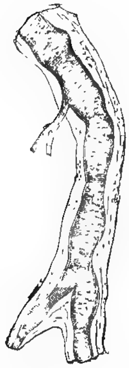
Fig. 126.



Miliary aneurism of very small vessels partly filled with blood. (Obersteiner.)

Predisposing Causes.—The nervous system may be in a condition of impaired resistance, and, this *predisposition* being present, various accidents and incidents may serve as *exciting* causes of nervous diseases. Among the most important predisposing causes are heredity, developmental influences, age, sex, race, occupation, habits, social condition, climate, poisons, special diatheses, and infection. A new significance has been given to predisposition by bacterial research. It may mean some slight difference in the chemical composition of the body, the excess of some element which is obnoxious to the bacteria, or the deficiency of some substance which is necessary for their vigorous growth; but it may mean only a greater or less vitality and resisting power on the part of the cellular and tissue elements. It may be local as well as general. (Cheyne.) Among the causes which lower the resistance of the nervous system are hereditary weaknesses in special directions, the presence of other poisons in the nervous system, the effects of trauma, and a poorly acting vasomotor system. (Putnam.)

Fig. 127.



Beaded enlargement of a large artery in the brain. (Obersteiner.)

Exciting Causes.—Some of the causes given as predisposing may also act as exciting influences in the immediate production of nervous diseases; and additional exciting causes of considerable importance are emotion, fright, mental strain, injuries, and reflex irritation. Emotion, fright, and psychical causes in general, as a rule,

produce functional nervous affections, but occasionally may be the starting point of organic disease, as of an attack of cerebral congestion or hemorrhage. Traumatisms need brief special consideration. Crushing, bruising, laceration, or division of nerves may occur; the brain and spinal cord may suffer from the effects of fracture of their bony encasements, or from hemorrhage on their surface or within their substance; vertebral dislocations may compress and crush the spinal cord; and the nervous system may suffer in various other ways in consequence of injuries. Traumatic nervous affections of great medicolegal interest result from railroad and other accidents, and here the influence of fright and general shock is often added to the direct effects of injury, while suggestion and self-concentration may also be etiological factors. Intracranial tumors are frequently ascribed to traumatisms, a contusion or local inflammatory lesion being produced and acting as a nest for the development of the growth. Not a few nervous affections are referred to reflex irritation of nerves in various parts of the body, as to genital, nasal, pharyngeal, ocular, ovarian, and uterine irritation.

Heredity.—The influence of dissimilar heredity is seen in functional affections, such as hysteria, neurasthenia, mania, and melancholia, which often appear in families with a history of alcoholism, epilepsy, syphilis, or tuberculosis. Epileptics and alcoholics have often inherited weak nervous systems from a pathological ancestry, and insanity, deaf-mutism, pauperism, and criminality are frequently results of atavism. Even acquired nervous affections can be inherited. Brown-Séquard produced in guinea pigs an epilepsy which became hereditary in subsequent generations of these animals. The term *neuropathic constitution* describes a diathesis manifesting itself in nervous and mental instability, which is not infrequently the inheritance of a condition acquired in a near generation. Strictly speaking, it is often the tendency to nervous disease rather than the disease itself that is inherited; it is the neurotic defect or neuropathic constitution which is handed down. The tendency to a recurrence of particular pathological types may continue through several generations, as is illustrated by the growing list of "family diseases," such as Friedreich's ataxia, hereditary chorea, muscular atrophy, and forms of spastic disorder. These affections are embryonal.

Developmental Influences.—Developmental influences play an important part in the etiology of certain nervous diseases. During the embryonal or fetal period occur arrests and aberrations, which give rise to malformations and monstrosities, and to various forms of idiocy and imbecility. During the rapid growth of the brain between birth and seven years of age, convulsions, night or day terrors, transient delirium, ephemeral fever, meningitis, encephalitis, and poliomyelitis may occur; chorea, epilepsy, and family forms of disease appear between seven and thirteen; during

puberty and adolescence the same nervous disorders may arise as just before puberty, with the addition of others such as hystero-epilepsy, insanities, and sexual perversions. "A man is as much a part of his ancestry, and his posterity is of him, as the root and stem are parts of one tree. The philosophic view of reproduction is that it is but one incident in a continuous protoplasmic life. Concerning hereditary defects and looking to the influence of nerve over nutrition, it seems reasonable to attribute early formative failures and the malformation of body and limbs, in some degree, to trophic innervation." The nerve cell develops its function with great slowness after its full bulk has been attained; and it is a rule with few exceptions that the tissues that are of slow development are most influenced by hereditary evil tendencies. (Clouston.)

Age.—The most frequent nervous diseases of infancy and early childhood are those which indicate unstable and irritable conditions of the nerve centres, such as spasms, head-banging, night terrors, ephemeral fever, and chorea. Diseases due to destructive central lesions are not common. Reflex irritative disorders in young children occur as the result of teething, and from digestive disturbances. Family affections appear at various developmental periods. Functional nervous disorders such as neurasthenia are most prevalent from adolescence to middle life. Hemorrhages into various parts of the nervous system, and especially into the brain, and degenerative diseases of all parts of the neuraxis, are, from their very nature, likely to occur beyond middle life. When they occur earlier they may be evidences of premature senility; as age in such cases is not to be measured so much by years as by the condition of the bloodvessels and nerve cells.

Sex.—The female sex is more liable than the male to certain functional nervous affections, as hysteria and neurasthenia, and to certain types of sensory disease, as headache, spineache, neuralgias, and migraine. Motor paralyses occur in both sexes, but are of greater frequency among boys and men. Types of disease vary according to the inherent powers and requirements of the two sexes, and according to differences in occupation and exposure. While the uterus and its appendages play a part in the production of nervous diseases, their influence has been overrated.

Race.—Special types of hysteria are determined largely by racial characteristics. Hystero-epilepsy and other forms of grave hysteria are most common among the Latin races. Melancholia and a special tendency to suicide are most prevalent in some of the German nations. Locomotor ataxia is of infrequent occurrence among negroes, although cases have been reported by Burr and others. The negro race suffers unusually from cerebrospinal meningitis, as has been evidenced in many epidemics in the Southern States in this country. The disease is more severe in the dark races than

in the white, the ratio of susceptibility being exactly in proportion to the depth of color, the blackest suffering most. Trismus neonatorum also has a high rate of mortality among negroes; and it is more common among Jewish and Mohammedan children than among those of other races. The Semitic people are prone to melancholia. (Byers.)

Occupation.—Occupation also influences the development of nervous diseases of certain types. Affections occurring among clerks, telegraphers, typewriters, artists, pianists, and others, have been erected into a class of occupation neuroses. Paralysis, tremor, and spasm are found among workers in lead, mercury, or arsenic. Headaches arise from work which causes eyestrain, and sleeplessness from too intense literary labor. Idleness is also a not infrequent cause of functional nervous disorder, and particularly of some types of hysteria and hypochondria.

Habits and Social Conditions.—The abuse of alcohol produces special types of acute and chronic nervous disease, and its effects are felt in the weakened nervous system of succeeding generations. The excessive use of tobacco has also an injurious effect upon the nervous system. Bremer holds that the use of tobacco in the young is productive of both mental and moral deterioration. Sexual abuses and irregularities have depressing psychical and physical effects, and if excessive may cause functional and organic affections, although their efficiency in the production of the latter has probably been overestimated. Vicious habits of psychical origin sometimes act as causes of nervous and mental disease: thus, hysterical anorexia may become established through the patient having feelings of discomfort which lead to the reduction of diet, and fixed delusions as to food. Muscular twitching, whether of spontaneous origin or the result of imitation, may become a habit chorea. Bad educational methods count for much in the causation of nervous disorders in growing children, and the complicated conditions and harassments of a high civilization, involving great mental strain, have a tendency to affect the nervous system injuriously.

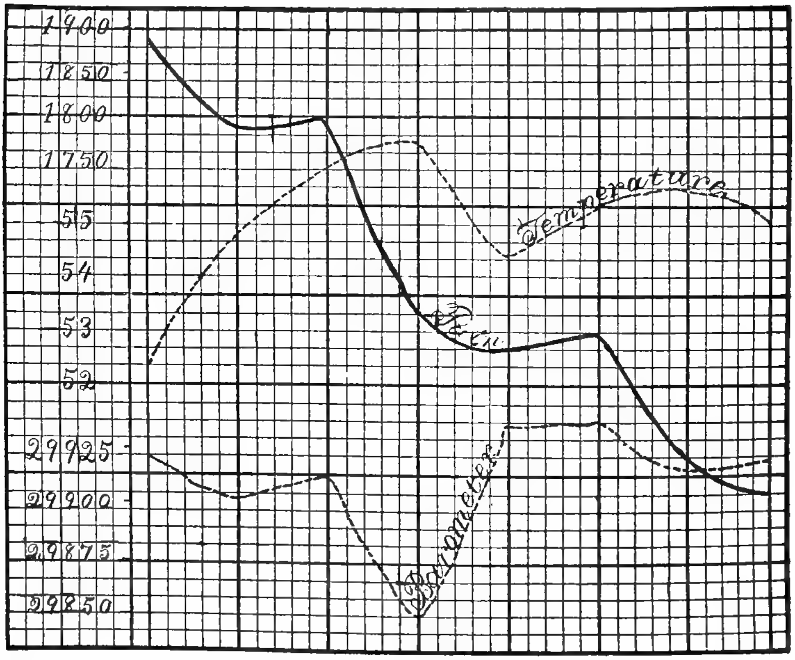
Climate.—Valuable data with reference to the influence of climate on nervous disease have been furnished by Eskridge, Solly, Curtin, Loomis, Reed, Hinsdale, and other members of the American Climatological Association. Some nervous affections, and especially neurasthenia and neuralgia, may be the result of a lack of equability in climate. The effects of altitude upon nervous patients are important. The high altitudes of Colorado, North Carolina, New Mexico, and other regions often resorted to for health, may be a disadvantage or a source of danger. Some affections which have been attributed to high altitudes are hysteria, general nervousness, melancholia, chorea, tachycardia, irregular heart, and apoplectiform attacks. Both blood pressure and the rhythm of the heart may be

injuriously influenced through the nervous mechanism of the vascular system. Besides the rarefied air, dryness, dust, wind, and an atmosphere highly charged by electricity affect nervous cases unpleasantly. Careful acclimatization is of the utmost importance. (Eskridge.) A dry climate, by diminishing the water in the blood, acts as a powerful stimulant to the nervous system, increasing its functional activity and causing excitement and sleeplessness; hence the healthy removed to a dry climate or to one of considerable elevation become restless. Nervous affections which are not absolutely initiated by climatic conditions may be aggravated by them.

Seasonal Influences.—In the spring, either as the result of severe and prolonged labor or as the effect of cold and exposure, the nervous system becomes lowered in tone, and in the weak and those predisposed by neuropathic constitutions, certain functional nervous diseases, as chorea, hysteria, and neurasthenia, develop. A large proportion of cases of chorea occur in the spring.

Weather.—Weir Mitchell and Captain R. Catlin, the latter a sufferer from "stump" neuralgia, have made important observations on

FIG. 128.

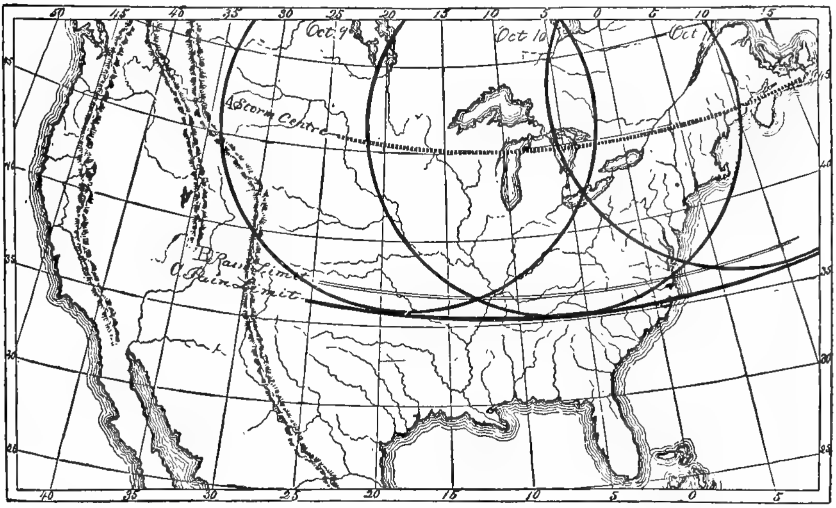


Annual pain barometer and temperature curves, 1875–1882 inclusive. (Mitchell and Catlin).

the relations of pain to weather. Less than half of the cases studied felt unusual sensations upon the coming of an east wind and during its continuance; and of these two thirds insisted on their power to predict such change in the weather, while the rest thought that any

great change was likely to cause them pain. An annual neuralgia curve was constructed on the monthly ordinates of neuralgic duration, as shown in Fig. 128; and a large number of neuralgic attacks were seen to be definitely related to storms, but no single element of mischief was discovered, and Mitchell believed that some combination of weather started the pain. At the centre of the vast rain area of every storm is a moving space of the greatest barometric depression, known as the storm centre, which the rain usually precedes by five hundred and fifty to six hundred miles, and before and around the rain is a forerunning neuralgic belt. Fig. 129 is a diagram of a storm with its theoretical rain area and neuralgic belt.

FIG. 129.



Relation of pain area to rain area. (Mitchell.)

Metallic and Gaseous Poisons.—Important central and peripheral nervous diseases result from the ingestion of metallic poisons, such as arsenic, mercury, lead, copper, zinc, and antimony; or from inhaling gaseous substances, such as carbon disulphide and carbonic acid. In carbonic acid poisoning central lesions are caused by the extreme congestion of the brain, the result of the direct action of the gas upon the vessels, with an undiminished action of the heart. (Schwerin.) Various poisons cause affections much like the scleroses. The symptoms of alum poisoning, as pointed out by Mayer and Siem, are like those of bulbar paralysis.

Diatheses.—Arthritic patients are often neurasthenic, insomnic, hypochondriac, or melancholic. Renal work is increased by arthritism, giving rise to toxins which may poison the central or the peripheral nervous system. A great stimulus to the study of the part played by uric acid in the production of various nervous and

mental disorders was given by the investigations of Haig, although long before his publications Maudsley advocated the theory of auto-intoxication by uric acid as a cause of melancholia. The blood of some cases of melancholia shows both a relative and an absolute increase in uric acid. Oxaluria is sometimes described as a cause of nervous disease, but how far it is a concomitant and how far a cause has not been satisfactorily determined. The rheumatic and gouty diathesis, and that form of half gout described by Da Costa as lithemia, produce distressing nervous symptoms, and a distinct type of lithemic neurasthenia has been erected by some authors. Gout in one generation may cause various forms of nervous disorder in succeeding generations.

Microbic Origin of Nervous Disease.—Microorganisms, only a portion of which are definable by the microscope, abound everywhere, and diseases of the nervous system are being more and more recognized as of bacterial origin, and particularly as due to certain bacilli and micrococci. Just how these bacteria produce their effects upon the nervous system is a question which has received much consideration. They may act directly, but in this case susceptibility must be great and the pathogenic bacterial dose must be large; commonly their effects are indirect, due to the toxins excreted by the bacteria. In some instances the bacteria and the chemical poisons which they excrete act together. In a restricted sense an "infectious disease" is produced by the direct influence of bacteria introduced into the body; bacterial toxic diseases are caused by the poisons secreted by microorganisms. In tetanus the active poison is generated at the place of its introduction and is gradually carried through the body, causing a general intoxication; but the toxin artificially manufactured and injected will cause the same phenomena as the direct tetanus infection. Even the protozoa, which are not true bacteria, probably play a part in the pathogenesis of a few nervous affections, as when malarial organisms exert their poisonous influence upon the central or the peripheral nervous system. Many organic nervous diseases are perhaps best regarded as due to protoplasmic poisoning, the toxin or infection having had a special affinity for nerve protoplasm.

Ptomaines and Leucomaines.—The term *ptomaine*, derived from the Greek word meaning a "corpse," is applied to all the products of the decomposition of organic bodies under the influence of microorganisms. What Gautier first termed *leucomaines* are basic alkaloidal substances which occur in living tissues as the result of physiological or pathological processes. It would perhaps be better, as has been suggested by Leffmann, to disregard both expressions and speak only of *basic nitrogenous bodies*. Such bodies are found in plants and animals; they are constantly produced by the most minute forms of life. These products are stored in the

structure of plants, and often give them their poisonous and their medicinal properties. Microorganisms and the higher animals, owing to the media in which the former live and the excretory organs with which the latter are provided, are largely able to rid themselves of these products, which would otherwise be injurious or even fatal.

Chemical and other Effects caused by Microbic and Toxic Agencies.—Some clear general idea should be had of the train of physical and chemical processes occurring from infection or poisoning in an organic affection of the nervous system. As a result of the action of microorganisms, ptomaines are excreted, and by their action upon the protoplasm of nervous tissues, and of other tissues, leucomaines are produced, and these may have a greater or less toxic influence in conditions of the system in which they are not properly excreted, thus giving rise to disease. The physician confronted by chronic, and even in some cases by acute, nervous affections of microbic origin has to deal not with the originating microorganisms, but with their primary or secondary products; or perhaps not with these, but with their effects upon nervous tissue—with the blurs and scars which they have left. The thoughtful neurologist must always bear such facts in mind. The same truths hold good for inorganic poisons, such as mercury, arsenic, and lead, which early give rise to structural nervous disease, which frequently after certain stages have passed does not improve under eliminative therapeutics because the pathological condition is dependent not upon the presence of the original toxic bodies, but upon deranged molecular states, resulting from the original agents.

Nervous and Mental Diseases due to Infectious Processes.—The infectious diseases admitted to be causes of nervous affections are tetanus, rabies, syphilis, tuberculosis, diphtheria, lepra, gonorrhea, typhoid fever, erysipelas, influenza, mumps, the acute exanthemata, the pyogenic organisms, the diplococcus lanceolatus, malaria, and actinomycosis. The diseases of the nervous system which are suspected but not fully proved to be of infectious origin are beriberi, poliomyelitis, Landry's disease; certain forms of myositis, neuritis, and myelitis; some of the cerebral palsies of children; chorea, epilepsy, disseminated sclerosis, and other cerebrospinal and spinal scleroses; amputation neuritis and herpes zoster. (Putnam.) Mental manifestations and even special types of insanity may be due to such causes as traumatism, anemia, exhaustion, and emotional shock, but they are also sometimes dependent on the action of infectious intoxication and probably on specific microorganisms, although the last cannot be regarded as absolutely proved. Febrile delirium and postfebrile insanity have been especially considered. A special type of confusional insanity has been suggested as following infectious and diathetic disorders, and this type is probably a toxemia and

due to a special poison. (Korsakoff, Hurd.) Numerous cases have been reported. Much negative evidence in favor of the infectious origin of acute mania or acute delirium has been furnished by clinico-pathological observations, autopsies revealing nothing positive. Various microbic agents are apparently capable of producing the same nervous or mental diseases.

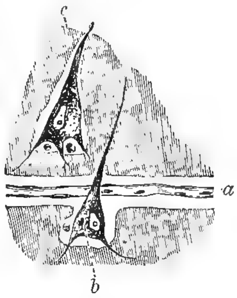
Syphilis.—As syphilis is one of the most frequent of the infectious causes of nervous disease, it requires a few words of special discussion. It affects either the central or the peripheral nervous system, acting both directly and indirectly, both recently and remotely, and both as an intrinsic and an extrinsic cause. Many so-called syphilitic affections of the nervous system are in reality diseases of its envelopes or of its vessels, as meningitis and arteritis; or the effects of such diseases, as when the brain substance is destroyed by hemorrhage or vascular occlusion. One of the most common forms of intracranial syphilis is the diffuse basal gummatous meningitis. The degeneration of nerves is often dependent on infiltrations from the epineurium. Tabes and the scleroses in general usually occur in syphilitic subjects, and syphilis is the most important of the toxic agents which can produce degenerative nervous diseases; and yet such affections are not curable by antisyphilitics, because what is present is a degenerative process, not the virus by which it has been initiated. Endarteritis obliterans, an acute or subacute inflammatory process which involves both the intima and the endothelial lining of arteries, is the result of syphilitic endarteritis, and especially affects the cerebral arteries, more often the vessels of medium size. It gives rise to many of the extrinsic affections of the brain, of the cord, and even of the nerves. Periarteritis, which frequently attacks the small arteries of the brain, causing miliary aneurisms and cerebral hemorrhage, is also often syphilitic.

Autoinfection and Autointoxication.—Functional nervous affections apparently directly related to gastrointestinal, hepatic, renal, and other conditions are probably due to poisons which are produced in the system by the diseases of these organs. Many processes result in disintegrations and reconstructions of poisonous materials which are carried through the system; and the retention of substances which, although normal in themselves, should in regular process be largely excreted, gives rise to certain nervous and mental symptoms and forms of disease. According to Albertoni and others, poisons in large quantities are formed by the action on food of the putrefactive bacteria of the intestines. Under most conditions this process is not pathological, but in indigestion excessive quantities of aromatic, albuminoid, and nitrogenous bodies, besides fatty acids, are formed. An old idea has been given a new dress by Klippel, who holds that hepatic disorder may not only maintain and exaggerate a psychosis,

but may be its true cause, a position which he has endeavored to establish both by clinical and by experimental investigations, adducing as evidence the presence of glycosuria and of certain hepatic coloring matters in the blood, also the decrease of urea and increase of uric acid. Urobilinuria present in these cases affords a method of testing the question. Numerous nervous and mental disorders are present during pregnancy and attend the puerperium. They are usually dependent upon the surroundings of the puerperal woman, or carelessness on the part of the attendants, but auto-infection has been advanced as an explanation in cases which occur when every precaution has been taken. During pregnancy, the production of toxins in the system is sometimes the cause of nervous and mental symptoms, such as headache, general nervousness, depression, nervous exhaustion, melancholia, and mania. The chorea of pregnancy, with its accompanying mental phenomena, is probably toxic in origin.

Phagocytosis.—The phagocytes or leucocytes are groups of cells which have the power of eating or taking into their substance solid particles, as the colorless corpuscles of blood and mucus, and the connective tissue cells. They are scavengers of the human body constantly at work. Metchnikoff, to whom the doctrine of phagocytosis is due, has likened specific inflammation to a warfare in which the invading army is represented by microorganisms, and the resisting force by the leucocytes. In certain conditions of disease of the brain and nervous system Deiters's or spider cells abound; and, according to Bevan Lewis, these abundant connective elements of the brain take an active share in the pathogenesis of mental decadence. In paretic dementia, in chronic alcoholism, in senile atrophy of the brain, and in some other mental and nervous affections, the true nerve elements, according to Lewis, are destroyed by these abundant scavenger elements. Septic intoxications are due to the effects produced by nonparasitic bacteria. When such bacteria are injected into the tissues a struggle follows between them and the cellular elements; and according as the leucocytes or the bacteria obtain the mastery will be the result for the patient. The doctrines of Metchnikoff and Lewis have not been generally accepted. Nuttall has shown that the destruction of virulent bacteria in the blood by leucocytes is not essential. Others hold that the germicidal power of the blood resides in the serum alone, and that phagocytosis is but a secondary process.

FIG. 130.



Section from the cornu ammonis showing perivascular and pericellular spaces: *a*, capillary vessel in a perivascular lymph space; *b*, pericellular lymph space directly continuous with the former: two leucocytes are seen in the pericellular space *c*, and one in the space *b*. (Obersteiner.)

SYMPTOMATOLOGY AND METHODS OF INVESTIGATION.

General Method of Study.—The same procedures may have their application in numerous affections differing widely in pathology, but general rules and principles will serve to guide in an investigation of disease of the nervous system. The examination will vary somewhat with the views and habits of the investigator, and sometimes according to the special object to be attained. It is a good practical method for most cases to obtain (1) statistical data, such as name, age, birthplace, and occupation; (2) an account of the onset of the disease; (3) its etiology, including family history; (4) a history of the patient from the onset to the time of examination; and (5) his present condition. The examiner may prefer first to obtain the family history and general etiology; but, as a rule, it is best to fix accurately the date of the onset of the illness, as other investigations will radiate naturally from this point, and it is not always an easy task to do this. The ataxic may fix as the beginning of his disease the time when symptoms of incoordination were first manifested, although he may have had for months and even years lancinating pains or affections of the eyes or of the bladder. When the time of onset is settled, inquiries should be made as to heredity; as to diseases of infancy, childhood, and youth; as to syphilis, alcoholism, exposure, injury, overwork, poisoning, or anything else bearing on causation. The period from the onset to the examination may cover hours or days, or may extend over months and years.

Classes of Symptoms.—In studying the present condition it may be necessary to investigate (1) psychical symptoms; (2) somatic stigmata; (3) disturbances of general sensibility; (4) morbid phenomena of the special senses; (5) motor disturbances; (6) reflex disorders; (7) electrical conditions of nerve and muscle; (8) vasomotor, secretory, and trophic symptoms; (9) symptoms of varying character referable to the great viscera of the body. Only one set of manifestations may be present in a case, while sometimes, as in tabes dorsalis or in dementia paralytica, sooner or later in the progress of the disease a long train of phenomena, covering almost the whole field of symptomatology, may appear.

Mental Examination.—The mental examination of a patient supposed to be insane is of first importance, but in nervous cases in general the study of psychic phenomena is also essential. In hysteria, neurasthenia, and the traumatic neuroses, the mental condition is often the most important factor; and in some organic diseases, as the hereditary choreas, scleroses, dystrophies, and chronic inflammatory diseases, mental disease may sooner or later appear. The psychical study must include a consideration of depression or exaltation, of delirium, violence, apathy, torpor, stupor, coherence, hallucinations, illusions, and delusions; of special tendencies—suicidal,

homicidal, destructive, or criminal; of automatic or cataleptic phenomena; of peculiarities of speech, and of powers of memory, judgment, comparison, and all the higher faculties. Instruments for the study of the psychology of the senses have been much added to in recent years, and these vary in number and complexity according to the fineness of the work to be done. It is of course not feasible for the physician in his daily work to use all the apparatus employed in the laboratory of the experimental psychologist, but some of the appliances of experimental psychology will be found practicable.

Time Relations of Mental Phenomena.—A fundamental determination in psychophysics was that of Helmholtz of the rapidity of nerve transmission. Important experiments have been made recently to determine the reaction time for the various senses both in the sane and in the insane. By reaction time is meant the time taken by a sense to respond to a given stimulus, which, according to James, is a pure reflex, not a psychic, act. "In the simple time reaction there is a physiological and also a psychological portion. The physiological elements include: first, the time for the sense reaction to respond to the impression; second, the time for the passage of the impulse inward along the nerves; third, the return passage of the motor impulse from the brain to nerve and muscle; fourth, the time for the contraction of the muscle. The time thus left unaccounted for is taken up by the psychological processes and transformation of the sensory into the motor impulse. The reflex act takes less time than the voluntary one. The reaction time differs for the different senses. Hearing is shortest, touch is intermediate, and sight longest. Reaction to the sense of temperature is longer than to contact. Reaction to heat is longer than to cold. When the stimulus is intense the reaction time is shortened. When the subject knows the nature of the experiment, and expects certain results, the reaction time is shortened." (Jastrow.)

Subjective Symptoms.—To give subjective symptoms their just value will often severely tax the diagnostician. They may constitute the entire case, or they may play an important coordinate or subordinate part; or the assertion of their presence may be indicative either of fraud or of self-deception on the part of the patient. It must not be concluded that because symptoms are subjective they are not real. Intellectual conditions often complained of are loss of the power of attention, melancholy, apathy, indifference, irritability, and emotionality; uncontrollable thoughts, doubts, and fears; tinnitus and partial or complete deafness; photophobia and eye pains; all sorts of paresis and paresthesia; twitchings and jerkings; and feebleness in standing and walking. It is always important to discriminate as to the real nature of pain. It may be hallucinatory, but even when not it may be cerebrally initiated. According to Raggi, pain reflexes following hallucinations may acquire an inten-

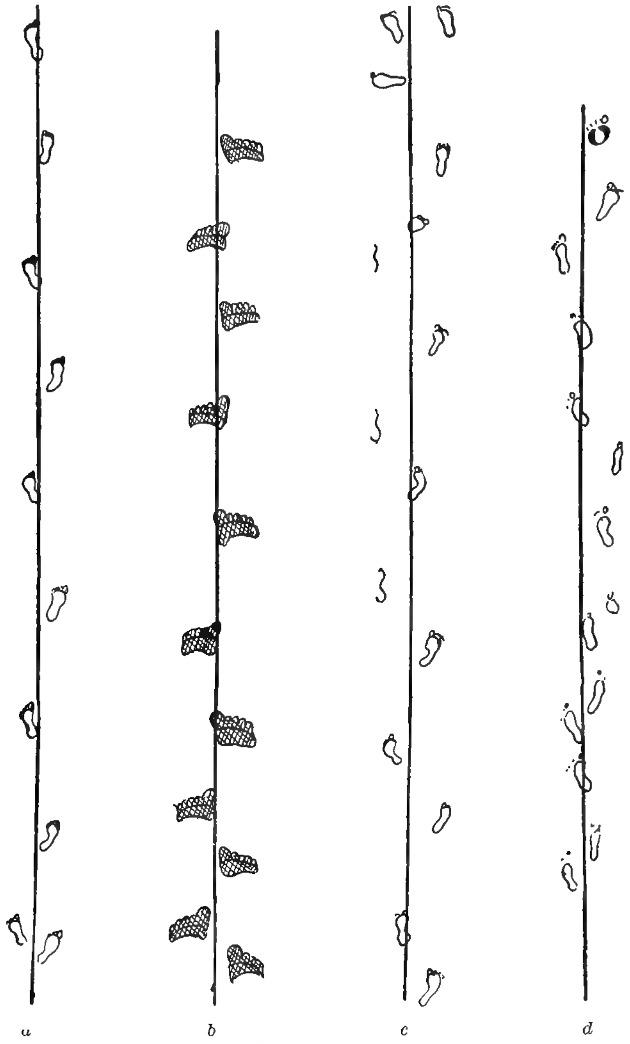
sity equal to those of true perceptive origin, and pain due to intellectual processes is a phenomenon of insanity. The subconscious pain of the hysterical is fundamentally delusional. Pain of cerebral origin due to organic lesion must be recognized. Mann has recorded a case of localized burning pain apparently caused by a lesion irritating the intraencephalic sensory fibres. Edinger has also reported a similar case in which the pain became so intense that the patient committed suicide. The autopsy showed softening of the external nucleus of the thalamus, the pulvinar, and a small segment of the internal capsule. The character of the pain described by a patient, whether aching, burning, darting, lancinating, fulgurant, intermittent, or constant, is often of diagnostic value, and a close cross-examination may be necessary in arriving at a conclusion. Other sensory phenomena besides pain may be of cerebral origin, as the brain itch described by Bremer. Many of the abnormal head sensations, such as drawing, compressing, weighing down, and crawling, of which nervous patients complain, are doubtless due to similar cortical processes, and here might also be placed the subjective false sensations of cold, described by Mitchell and others. Many nervous patients suffer from forms of tinnitus, the cause of which must be sought through an examination of the ears and hearing, of the cerebral symptoms, and of the general condition of the patient. Among the subjective visual disturbances are photophobia or fear of light, photopsia or sensations of phosphenes, luminous circles or sheets, sparks, flashes, balls of fire, colored lights, fortification lines, polyopia or multiplied sight, and muscæ volitantes or flying bodies.

Inspection.—Many signs of nervous disease can be noted by careful inspection. The gait may be seen to be notably spastic, ataxic, or paretic. A diagnosis can often be made in hemiplegia, sclerosis, or hysteria, by a study of either the carriage or the gait. Fig. 131 compares the normal gait with that of disseminated sclerosis. The slow, grotesque, mobile spasm of athetosis, and the different forms of chorea and tremor, are studied chiefly by sight. Fibrillary tremor may be observed in the muscles anywhere, and is sometimes strikingly exhibited in the tongue. Vasomotor affections reveal themselves to the eye by flushing, pallor, or irregularities of color, as well as by differences of temperature to the hand and to instruments. The complexion and morbid changes in the skin due to trophic disease are to be studied in the same manner. The facial expression in melancholia, in mania, in various forms of idiocy and imbecility, and in neurasthenic and hysteric patients is often distinctive. Opposite mental states give opposite facial signs.

Stigmata.—Somatic stigmata are the landmarks or body marks of physical degeneration, the evidences of arrested or irregular development; they include differences in stature and in the length

of limbs; irregularity, undue symmetry, or abnormal shapes of the skull or face; deformities of the tongue, palate, ear, eye, or genitals: in brief, constitutional deformities in general. Stigmata may

Fig. 131.



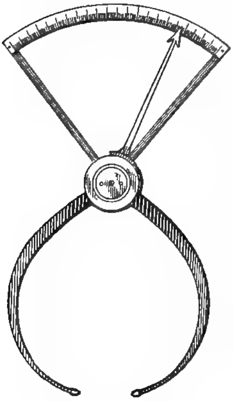
Normal gait compared with the gait of disseminated sclerosis: *a*, normal gait, the spaces are equal, and the feet follow the same line of direction; *b*, disseminated sclerosis, steps short, only the front of the foot applied to the ground, toes turned under, tendency to encroach on the median line; *c*, disseminated sclerosis (cerebellar gait), steps unequal in length, direction irregular; *d*, disseminated sclerosis (cerebellar gait), same as *c*, with greater irregularity. (Marie, after Gilles de la Tourette.)

be present in the normal, but they occur in a large percentage of the idiotic, insane, and criminal. Anesthesia, analgesia, hyperesthesia, hysterogenic zones, changes in the visual field, transferred

sensations, and various vasomotor and trophic phenomena are often spoken of as hysteric or psychic stigmata.

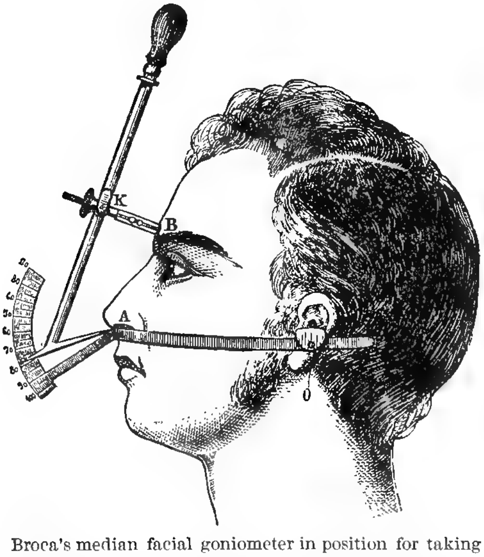
Cranial Investigations.—Cranial investigations are concerned (1) with the examination of the heads and skulls of different races, of the idiotic and the insane, of infantile paralytic and spastic cases, and of those showing deformities due to disease or accident; (2) with examination to determine as to the necessity of surgical operation or its site, which will be considered under cranial topography. Marked peculiarity of cranial configuration may indicate development or lack of development or disease of lobes or lobules, and even sometimes of gyres. The parietal eminence corresponds to the cortical motor zone; the forehead and front of the head bear some relation to the development of the prefrontal lobes, and prominences in the occipital and temporal regions to the visual and auditory areas. Cranial asymmetry is most strikingly manifest in many cases of infantile spastic hemiplegia, a subject to which Peterson and Fisher have given special attention. For measurements of the skull it is necessary to have (1) a tape line or ribbon, which should be marked on at least one side in the metric system; (2) calipers or compasses with curved legs (Fig. 132); (3) a goniometer (Fig. 133). Strips of lead which adapt themselves readily to the shape of the head may also be used, and interesting examinations can be made by the hat conformateur, or by an instrument devised by Luys, based on the same principle. The thickness and arrangement of the hair must be taken into consideration. It is always best to have the hair closely cut. The greatest care must be taken to mark accurately corresponding points

Fig. 132.



Benedikt's calipers.

Fig. 133.

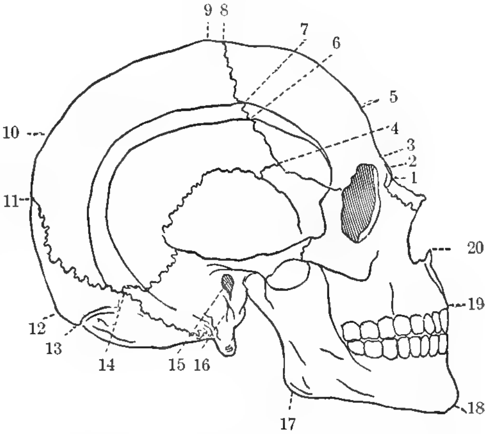


Broca's median facial goniometer in position for taking the facial angle, whose apex is at the subnasal point: the two auricular pins O are in place, A is at the superior alveolar point (subnasion), and the branch K B is in its proper position. (Topinard.)

on both sides of the head, and not to allow the tape to slip during the measurement.

Skull Landmarks.—Certain skull landmarks, indicated in Fig. 134, have been fixed and named by Broca, and will be found useful. The *nasion* is the junction of the nasal and frontal bones. The *glabella* or *glabellum* is the triangular space between the eyebrows. The *ophryon* is on a level with the superior border of the eyebrows, and corresponds nearly to the glabella. The *bregma* is the junction of the sagittal and coronal sutures; *vertex*, the superior point of the skull usually just caudad of the bregma; *obelion*, the part of the sagittal suture between the parietal foramina; *lambda*, the confluence of the sagittal and lambdoidal sutures; *inion*, the external occipital protuberance; *basion*, the middle of the anterior wall of the foramen magnum; *asterion*, the junction of the occipital, parietal, and temporal bones; *gonion*, the angle of the lower jaw; *stephanion*, or, better, *superior stephanion*, the intersection of the ridge for the temporal fascia and the coronal suture; *inferior stephanion*, the intersection of the ridge for the temporal muscles and the coronal suture; *metopion*, the median point between the two frontal eminences; *auricular point*, the centre of the orifice of the external auditory meatus; *pterion*, the junction of the great wing of the sphenoid and of the frontal, parietal, and squamous bones; *prosthion*, the alveolar point; *subnasion*, the subnasal point; *gnathion*, the lower jaw or mental point.

Fig. 134.

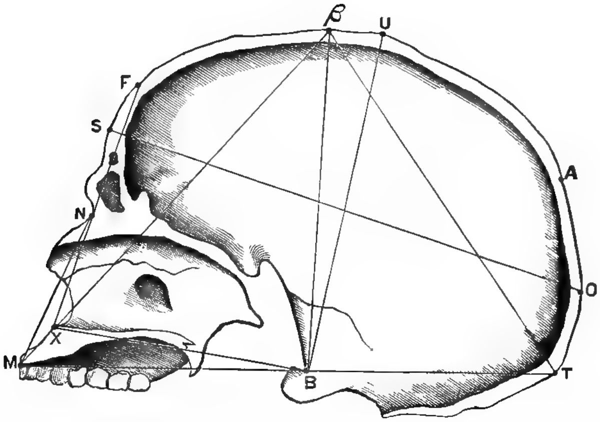


Skull landmarks as given by Broca: 1, nasion; 2, glabella; 3, ophryon; 4, pterion; 5, metopion; 6, inferior stephanion; 7, stephanion; 8, bregma; 9, vertex; 10, obelion; 11, lambda; 12, maximum occipital point; 13, inion; 14, asterion; 15, auricular point; 16, basion; 17, gonion; 18, gnathion; 19, prosthion; 20, subnasion.

Craniometrical Methods.—The following are definitions of the terms most commonly used: (1) *circumference*, a measurement usually taken horizontally around the glabella and the *maximum occipital point*, which is just above the external occipital protuberance; (2) *volume*, or cubic contents of the skull; (3) *nasooccipital arc*, from the root of the nose to the lowest occipital protuberance; (4) *nasobregmatic arc*, from the root of the nose to the bregma; (5) *bregmatolambdoid arc*, along the sagittal suture from the bregma to the lambda; (6) *binauricular arc*, from the centre of one auditory meatus

to the other across the top of the head; (7) *anteroposterior diameter*, from the glabella to the inion; (8) *greatest transverse diameter*, the greatest transverse diameter of the cranium wherever found; (9) *length-breadth index* or *cephalic index*, the breadth of a skull multiplied by 100 and divided by its length; (10) *binauricular diameter*, the diameter connecting the two external meatuses; (11) *auriculo-bregmatic radii*, lines projected on the cranium from the bregma to the auditory meatuses or auricular points, between the bregma and each meatus; (12) *facial length*, the distance from the root of the nose to the lowest part of the chin; (13) *empirical greatest height*, the approximate measurement of the distance between the basion and the vertex of the skull. The three natural planes of projection in the skull are, (1) the median plane, that is, the plane which con-

FIG. 135.



Craniometrical diagram: A, lambda; B, basion; MβT, triangle for ascertaining empirical greatest height (B to β or B to U); SO, anteroposterior diameter; BX, basal length of face; NX, length of nose; N to β, nasobregmatic arc; β to A, bregmatolambdoid arc; N to T, nasooccipital arc; F, frontal point or metopion; X, subnasion. (After Benedikt.)

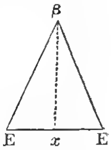
tains as many median anatomical points as possible; (2) the plane which passes through both visual axes of Broca and is perpendicular to the median plane; (3) the plane which is perpendicular to both the others and passes through each lateral point. (Benedikt.) The facial angle is the degree of obliquity of the face, but unfortunately the term has been used with different meanings. The facial angle of Broca is included between two lines drawn from the nasal spine to the ophryon and auricular point (auditory meatus), as shown in Figs. 133, 135.

Skull Measurements.—The following table, modified from tables by Peterson and Fisher, gives the craniometrical measurements of the normal adult skull, from which about six per cent. should be deducted for the hair and scalp, and compares with them those of twenty cases of hemiplegia spastica infantilis.

	Heads of Twenty Spastic Hemiplegics.			Normal Head.		
	Men's average (eleven cases).	Women's average (eight cases).	Boy (one case).	Average in adults, in centimetres. Males.	Females.	Physiological variation.
Circumference	53.7	52.3	47.0	55	53	51.5–60.4
Volume	1466	1281	1151	1500	1300	1201–1751
Nasooccipital arc	34.5	32.1	31.0	35.0	34.0	31.0–41.0
Nasobregmatic arc	12.6*	11.3	9.7	12.5	12.0	10.9–14.9
Bregmatolambdoid arc	12.2	12.1*	8.6	12.5	11.9	9.1–14.4
Binauricular arc	34.3	32.6	29.0	35.0	34.0	31.4–38.0
Anteroposterior diameter	18.2	17.9	16.3	18.7	18.2	17.5–20.0
Greatest transverse diameter	14.4	14.2	13.1	15.6	15.0	14.0–17.5
Length-breadth index	79.2	79.9	80.3	82.2	83.8	76.1–87.0
Binauricular diameter	12.0	11.4	10.1	13.1	12.6	11.6–14.6
Height, βx[1]	12.5*	11.6	11.1	12.2		10.5–13.1
Facial length	12.1	11.0	9.0	12.37	. .	10.5–14.4
Empirical greatest height	13.6	12.2	.	13.8	12.8	12.0–15.5

Sizes and Shapes of Heads.—*Microcephalic* technically is a term applied to heads less than seventeen inches in circumference; *brachycephalic* means broad and short headed, the length being to the breadth as 100 to 80, or more; *dolichocephalic* is long headed, the length being to the breadth as 100 to 75, or less; *mesocephalic* refers to a medium size between these. A *plagiocephalic* skull is one so out of shape that the features lie in an oblique plane; a *scaphocephalic* head is shaped like the keel of a boat turned upside down; *acrocephalic* or lofty, *trigonocephalic* or triangular, and *oxycephalic* or steeple shaped, are other descriptive terms sometimes used. The left side of the head is usually more developed than the right. Heads larger on the right than on the left, and heads of the same or nearly of the same dimensions on both sides, are present in large proportion among idiots. While unusual differences of the two sides of the head are evidences of abnormality, even-headedness is also a sign of low type. Left-headedness is common among the insane, and is more marked in some forms of insanity than in others. Skulls increase in size and change in shape as years advance up to late childhood. The female skull differs in some particulars from

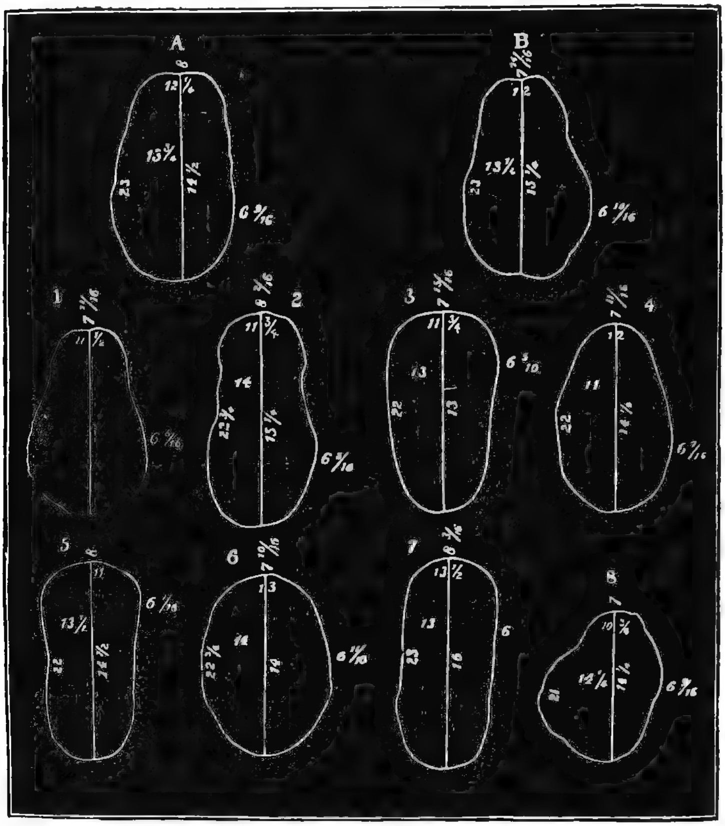
[1] The height βx of the triangle $E\beta E$, formed by the auriculo-bregmatic radii and the binauricular diameter.



* These averages are above the normal; all others are below the normal average.

that of the male. In Fig. 136 are outlines by Fletcher Beach showing different shapes of the head in idiocy, imbecility, and insanity.

FIG. 136.



Outlines of heads of idiots, imbecile, and insane: A, left-headedness in an insane person; B, right-headedness in an idiot. Shapes: 1, pyriform; 2, panduriform; 3, ovate; 4, obovate; 5, cuneiform; 6, rotund; 7, oblong; 8, irregular. (Beach and Clapham, in Tuke's Dictionary.)

Limb Measurement.—Limb measurements are sometimes almost as important in neurological as in surgical practice. Wight gives the following as errors which may arise from the attitude of the lower limbs. Abduction of the lower limb shortens its measurement, adduction lengthens its measurement: so that error will arise when one lower limb is adducted and the other abducted. In a case of this kind there would be a scientific error. Flexion of the lower limb shortens and extension lengthens its measurement. If the lower limb be abducted and flexed, and the other be adducted and extended, the error in measurement will be greater still. Tilting the pelvis acts like adduction and abduction. The normal asymmetry of the lower limbs is another source of error in their measurement, and still another is the fact that the lower limb can be elon-

gated by means of traction or pull, as was determined by Wight by experiments with a dynamometer. Thomas G. Morton has given close attention to the question of imperfect symmetry, especially with reference to inequality in the lengths of the lower limbs as a cause of curvature of the spine. He has described a new method and apparatus (illustrated in Figs. 137, 138) for readily and accurately determining any variation in the length of the lower extremities. The apparatus consists of a strong box, twelve

FIG. 137.

Morton's apparatus for the determination and measurement of asymmetry in the lower limbs: shortening of left leg shown.

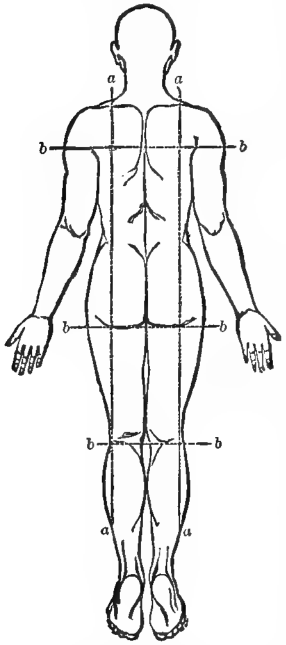
FIG. 138.

Morton's apparatus for the determination and measurement of asymmetry in the lower limbs: limbs symmetrized.

inches square, on the top of which are two movable foot-pieces or platforms, which can be projected upward by a screw acted upon by the handle, one on each side. On the edge of the platform, as it is elevated, is a measure to indicate the amount of shortening of the limb. If the line of the nates is vertical and the gluteofemoral line horizontal, shortening is not present, but if the line of the nates deviates, the horizontal gluteofemoral line will likewise deviate. If the deviation of the former is to the left, the

left gluteofemoral line is below that of the right, and the left limb is short (Fig. 137). If now the handle next to the left limb is made to elevate the left table or platform, so soon as the shortening is overcome the nates become vertical and the gluteofemoral line horizontal (Fig. 138), and the amount of shortening can be read upon the measure. Fig. 139 is an outline of the human back, showing normal vertical and horizontal lines for comparison. The question of asymmetry of the limbs is sometimes important in medicolegal work, especially in cases of alleged injury.

Fig. 139.



Outline of the human back, showing normal base vertical lines, *a a*, *a a*, and normal base horizontal lines, *b b*, *b b*, *b b*. (Morton.)

Sensory Disorders and their Terminology.—The term *anesthesia* covers all forms of sensation, or indicates only the loss of tactile sense, to which latter meaning it is best restricted. *Analgesia* is insensibility to pain. *Hyperesthesia* is exalted sensibility. *Thermoanesthesia* is loss of the temperature sense—of the power of detecting if a body is hot or if it is cold. *Hypercryalgesia* is abnormal sensitiveness to cold, and *hyperthermalgesia* abnormal sensitiveness to heat. In *dissociated anesthesia* one form of sensibility is lost and others are retained. Pain and temperature are more frequently abolished, or to a greater degree abolished, than touch. *Dysesthesia* is a feeling of distress or discomfort, and *paresthesia* describes morbid or perverted sensations. *Metamorphosis of sensation* may take place, as when the patient recognizes a prick as a burning sensation, or the reverse. A single prick or other stimulus may be perceived as several, this being *polyesthesia*. In *retarded* or *delayed sensation* the sensation experienced is separated by a distinct interval from the time of the application of the stimulus. A rough but often efficient method of determining retarded sensation is by touching or pricking the patient and noting by the second hand of a watch the amount of delay; but chronoscopes and chronographs can be used for scientific examinations. A *recalled sensation* is one alleged to be felt a considerable time after the patient has once received and responded to a stimulus. In *sensory tetanus* the patient cannot distinguish pricks if they are frequent. Under the name *allochiria*, Obersteiner first described a phenomenon in which sensations received on one side of the body were felt on the opposite side. The sensation may be felt not only on the opposite side but on both sides at the same

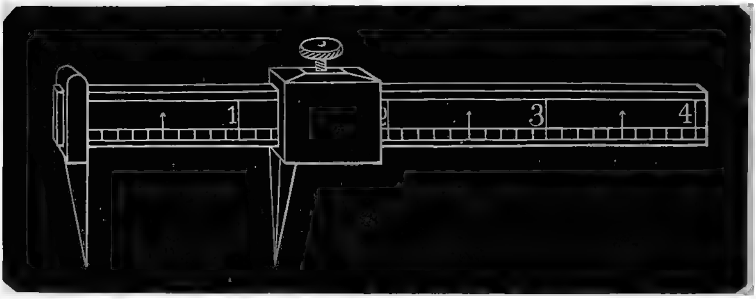
time. Allied to allochiria are other defects in the localization of sensation, as when a prick on one part of the limb is felt and referred by the patient to another, which is spoken of as *transferred* sensation. Gustatory, auditory, and visual allochiria have also been recorded. In color hearing, or *pseudochromesthesia*, and color gustation, or *pseudogeusesthesia*, two or more senses are simultaneously affected by the excitation of one. A certain sound of the voice, or a certain name, or the sound of an instrument, is interpreted by a particular color, as well as by sound. This phenomenon was first described by Verga in 1865. Mendoza has described phenomena allied to colored audition and colored gustation under the name of false secondary sensations or *pseudesthesias*. An actual perception of one sense gives rise to false perceptions of another or of other senses. The phenomena are sometimes described as *photisms*. An alphabetical photism is the visual image suggested by particular vowel or consonant sounds.

Sensory Investigations.—Important disturbances of sensation are easily overlooked by a careless or hurried investigator, or by one who does not understand the best methods of examination. The study of cutaneous sensations requires time, patience, and method. The work must be done carefully, or the results are worthless, particularly for tactile sensibility. The personal equation must be considered. The method of discriminating between two points, if very carefully applied, is sufficiently accurate for many cases, but often the results obtained are utterly worthless. The perfectly healthy may give diverse and confusing answers when tested with the esthesiometer. It is an error to decide that objective insensibility exists from the statements of the patients with reference to their feelings. Many patients refer to a paralyzed member, or a portion of it, as feeling heavy, or numb, or cold, or as asleep; but often in these cases careful and elaborate testing will fail to reveal true anesthesia. Cutaneous sensibility can be studied by touching, tickling, pinching, pricking, pressure, contact of hot or cold, hard or soft, rough, smooth, round or angular bodies, and analogous methods. A practical method of testing for sensation, recommended by Gilles de la Tourette, is to instruct the blindfolded patient to count one, two, three, etc., for each impression felt, so that if the anesthetic area is entered a count or counts will be omitted, and the reverse. Weber discovered the simple law that the increase of stimulus necessary to produce an increase of sensation bears a constant ratio to the total stimulus. An increase or diminution of pressure, for example, will be felt as soon as the added or subtracted weight amounts to one-third of the weight originally employed; a change in muscular sensation whenever the stimulus is increased or decreased by one-seventeenth; and a difference in a sensation of light when the increase or diminution is one one-hundredth. The powers of discrimination are much finer in some than in others, and the

ability to detect small variations is greatest in the intellectually ablest. Sensitivity is higher in men than in women. Men, as a rule, are employed as tuners of pianos, tasters of wine, and sorters of wool. Krohn, in experiments on simultaneous stimulations of the sense of touch, found that the skin of the joints is more sensitive than any other dermal locality; that touches on the back are better localized than on the front of the body; that localizations for dermal sensations are better for those on the median line than for those that are not; and that dermal sensations are more correctly localized on the right side of the body than on the left.

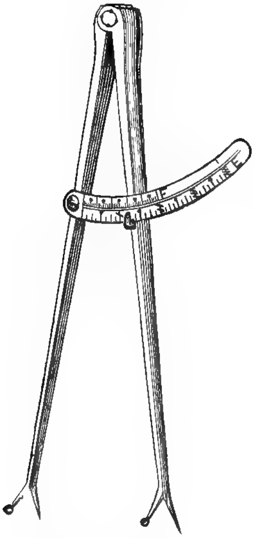
Esthesiometers.—Instruments for measuring sensibility are called esthesiometers. An esthesiometer of the form seen in Fig. 140 was suggested by Sieveking. Seguin had Sieveking's esthesiometer constructed of aluminium and of small size. Carroll's esthesiometer (Fig. 141) is bifurcated, and each arm has one bulb or blunt extremity, and one sharp point, so that the pain as well as the touch may be tested. The working of the instrument depends on the fact that in health we are able to distinguish two impressions made simultaneously on the skin only when these impressions are separated by certain distances, varying for different regions of the body. In using this instrument, the patient's eyes should be covered or he should be made to look away during the tests. He should be directed to speak at once as soon as the point or points are felt. If he stops to think about the matter, his answers often become worthless, as it is a question of instant recognition, and not of reasoning.

FIG. 140.



Sieveking's esthesiometer.

FIG. 141.



Carroll's esthesiometer.

Smallest Distance at which Points can be Distinguished as Double.—The following is a table of the smallest distances, in millimetres, at which two points of a pair of compasses can be distinguished as double by an adult, and by a boy twelve years of age. (Landois and Stirling.)

	Adult.	Boy of Twelve.
Tip of tongue	1.1 mm.	1.1 mm.
Third phalanx of finger, volar surface	2. —2.3	1.7
Red part of lip	4.5	3.9
Second phalanx of finger, volar surface	4. —4.5	3.9
First phalanx of finger, volar surface	5. —5.5	
Third phalanx of finger, dorsal surface	6.8	4.5
Tip of nose	6.8	4.5
Head of metacarpal bone, volar	5. —6.8	4.5
Ball of thumb	6.5—7.	
Ball of little finger	5.5—6.	
Centre of palm	8. —9.	
Tongue, white of lips, ball of thumb	9.	6.8
Third phalanx of great toe, plantar surface	11.3	6.8
Second phalanx of fingers, dorsal surface	11.3	9.
Back	11.3	9.
Eyelid	11.3	9.
Centre of hard palate	13.5	11.3
Lower third of forearm, volar surface	15.	
In front of zygoma	15.8	11.3
Plantar surface of great toe	15.8	9.
Inner surface of lip	20.3	13.5
Behind zygoma	22.6	15.8
Forehead	22.6	18.
Occiput	27.1	22.6
Back of hand	31.6	22.6
Under the chin	33.8	22.6
Vertex	33.8	22.6
Knee	36.1	31.6
Sacrum and gluteal region	44.6	33.8
Forearm and leg	45.1	33.8
Neck	54.1	36.1
Lower and middle dorsal and lumbar regions	54.1	
Middle of neck	67.7	
Upper arm, thigh, and centre of back	67.7	31.6 to 40.6

Special Methods of Studying Cutaneous Sensibility.—Oppenheim has suggested the method of touching symmetrical spots on both sides of the median line at once, and noting whether the patient appreciates the touch on both sides equally. Funke has proposed the use of solutions of glycerin in water. Drops of solutions of different densities are placed upon adjacent spots by the tips of the examiner's fingers and the patient is then called upon to decide which is the most viscid. The degree of disturbance is shown by noting the percentage of the two solutions which the patient declares to be different. An *algesimeter*, described by Hess,

consists of a metal tube with an arrangement for protruding a needle from its bottom to any extent desired. It has the advantage of furnishing a fixed unit of pain, which cannot be said of cruder methods. Motor sensations of the skin are determined by drawing a point up or down the skin and ascertaining whether the direction can be recognized. To test the sense of locality, let the patient close his eyes, then touch him on the forearm with a pencil point, and require him to touch the same point with another pencil immediately afterwards. The amount of error can be estimated in millimetres and averaged for a number of trials. (Sanford.) The often cited Aristotle's experiment has reference to this sense. It is to cross the middle finger over the first in such a way as to bring the tip of the middle finger on the thumb side of the first finger, and insert between the two a pea or other small object, when a more or less distinct sensation of *two* objects will result, especially when the fingers are moved.

Temperature Sense.—Objects of different temperatures but of the same size and of the same thermal conductivity should be applied to the skin. These may be small wooden cups with a metallic base, filled with warm and cold water, the temperature being regulated by a thermometer placed in the cups. (Nothnagel.) Hot and cold spoons, or absorbent cotton dipped in hot water or ice water, or test tubes filled with hot or cold water, may be used. Two objects, as thermometers with large bulbs, the temperature of each of which is known, may be applied successively to the same spot, and the patient required to distinguish between them. *Blocq's esthesiometer*, founded upon the suggestion of Nothnagel given above, has at the base a chamber with a flat surface to be applied to the skin, and this also has within it a thermometer which passes through its top. Heating it to different temperatures and applying it to the skin, the degree of temperature which the patient appreciates as heat or as cold is noted. The indifferent range where objects are felt to be neither warm nor cold is from about 80° to 85° F. A temperature as low as 62° should always be felt as cold, and one about 90° as hot. Sometimes response to the stimulus of heat is present, while that of cold is absent, or the reverse.

Muscular Sensibility.—Whether or not loss of muscular sense occurs without general anesthesia of the limbs, certain phenomena classed under "muscular sense" need to be studied. Cutaneous, articular, muscular, tendinous, and bone sensibility must enter more or less into the recognition of weight, resistance, pressure, and posture. The internal surfaces of the articular cartilages have a sensibility which is called into play by every movement. It is well known that joint surfaces may be extremely sensitive, as in ataxics. Muscular contraction, according to James, is only directly instrumental in giving us space perception by its effects on surfaces; and

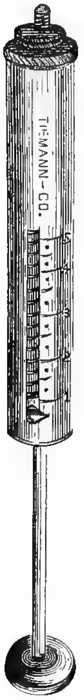
the intrinsic muscular sensations are likely therefore to be merely those of massive strain or fatigue, and to carry no active discrimination with them of lengths of the path moved through. Into muscular sensations enter the so-called *pressure sense*, the *sense of coarse movements of the limb*, and the *posture sense*, by which parts of the body are located. (Preston.) One difficulty in arriving at conclusions as to the muscular sense is that the test for it must depend in whole or in part upon a subjective state and the personal equation. In the investigation of the muscular sense, differences of surface, of temperature, and of the traction employed should be carefully considered or excluded. To exclude surface differences in testing by balancing weights, these can be suspended in a towel or by strings or cords. An experimental method used by Ferrier and Lauder Brunton is first to determine blindfolded the difference in weight which can be told by the hand held on a flat cushion and then to test the muscular discrimination of the same weight when the wrist is so flexed as to raise the weight with the fingers. By repeated experiments with weights varying from one to six ounces, it was found that the average discrimination by the sense of cutaneous pressure was one third, while the muscular discrimination accorded pretty nearly with one seventeenth. Repeating the same experiments with the same hand, a galvanic current was applied to the flexor muscles of the hand, so as to cause repeated raising of the weight of the fingers, when the sense of pressure averaged the normal, and again muscular discrimination was found to be almost as accurate as in former experiments.

The Pressure Sense.—A series of test weights consisting of a number of common gun cartridge cases filled with alternate layers of shot, wool, and wadding can be employed. The appearance of these is identical, and their weights form a geometric series. The testing is performed by handing to the person operated upon pairs of weights until his power of discrimination is approximately made out, and then proceeding more carefully. Care should be taken to exclude differences of temperature and prevent the displacement of the weights. (Galton.) For delicate testing of the sense of pressure, it is necessary to have bits of cork and very small weights for minimum pressure. The subject's arm and hand should be supported in a comfortable position with the palm upward, and a piece of blotting paper just large enough to prevent the weight from touching the skin should be placed in the palm. Starting with some standard weight, heavier and lighter objects should be used, and notes made of the power of discrimination in a large number of trials. Jastrow used for testing the pressure sense a modification of Fairbanks's post office balance, in which the initial and incremental weights were placed upon the scale pan, thus exerting an upward pressure upon the finger situated at the end of the beam.

Attachments were added by which the pressure could be constantly released from the finger and the ill effects of fatigue averted.

Piesmeter or Baresthesiometer.—An instrument called the baresthesiometer or piesmeter (Fig. 142) was invented by Eulenberg, and modified by Beard and Rockwell, for the determination of the individual sense of application of weight. This instrument consists of a cylinder about three-fourths of an inch in diameter and three inches long, in which is a piston which is kept pressed back to its fullest extent by a spring. At the end of the piston rod is a flat disk, which is placed against the skin of the patient. The operator then presses against the end of the cylinder, thus forcing the piston into the barrel, and the amount of pressure made before it is recognized by the patient is indicated by the scale on the instrument.

Fig. 142.



Beard and Rockwell's piesmeter.

The Posture Sense.—The posture sense is composed of afferent impulses derived from the skin, muscles, tendons, articulations and their coverings, and bone. Its loss is common in locomotor ataxia. In testing for it all the other senses should be excluded. The subject is blindfolded and told to touch certain parts of the body, as the nose, first with one finger and then with another; to put the two forefingers together; to place one hand on the other, after varied passive movements have been made with one arm by the operator; to put the heel of one foot on the toe of the other, and to imitate with one limb as exactly as possible the position of the corresponding limb, which has been arranged by the investigator. These experiments may be made reasonably exact, and may be greatly varied.

Sensations of Motion.—To Mach and Crum Brown we are indebted for much of our knowledge of the sensation of motion and its organ. A body can be moved in two ways, by translatory motion and by rotation. Translatory motion is always oriented in the same way, as when an object moves so that the same side always looks up or down and the same side looks always to the right or left. Rotational motion is that experienced when an object is turned round, although technically it is the change in motion or the acceleration which is perceived. The organ of this sense is the nervous apparatus of the utricle, which is that part of the membranous labyrinth of the semicircular canals which is lodged in the vestibule, and from which proceeds the vestibular nerve. It may be necessary to diagnosticate diseases of the semicircular canals and vestibular nerve and their terminations from affections of the cochlear

nerve, which is the true nerve of hearing. Often disease affects both the organ for the sensation of motion and the organ of hearing, owing to their close contiguity and the associations in function of this space sense and the auditory sense; but even peripheral affections of the sense of motion may be present without any disturbance of hearing. The patient should be first studied negatively, that is, by excluding disturbances of hearing, and next by the careful study of the accounts which he gives of his own sensations. Experiments for studying the sensation of rotation can be made with a rotation table, which for ordinary purposes, as suggested by Sanford, can be made by laying a board seven feet long across an ordinary turning chair or screw stool without a back. The patient is seated upon the board blindfolded, and the stool is turned evenly in one direction or the other. In health the approximate direction can be recognized when the rate is as slow as two degrees per second or even slower. The rotation can be increased in rapidity; the subject can be rotated for half a turn and then stopped suddenly; and other similar experiments can be performed. Differences in the effect of motion when made to the right or left, or when the head is held in various positions, may teach something as to the condition of the semicircular canals. Great care should be taken to avoid falls. A knowledge of the effects of such experiments upon a healthy subject is of course necessary. Sometimes the usual results will be entirely absent, as in some deaf mutes, or when the nervous apparatus of the vestibule and semicircular canals is entirely destroyed.

Visual and Ocular Disturbances.—Changes in the optic nerve and retina, such as optic neuritis and optic atrophy, are of the greatest importance in the study of affections of the brain, like tumor and meningitis, and these conditions and the affections which counterfeit them will receive attention under diseases of the optic nerve. Disorders of accommodation and refraction need to be part of a neurological examination, but details of examinations for such disturbances belong to the ophthalmologist. *Exophthalmus,* or protrusion of the eyeball, may be an indication of an affection of the gangliated system of nerves. *Nystagmus,* or involuntary oscillation of the eyes, usually horizontal or bilateral, is present in diseases of the cerebellum, disseminated sclerosis, and other nervous affections.

Hemiopia and Hemianopsia.—*Hemiopia* refers to loss of visual power in one-half of the retina, and *hemianopsia* to half loss in the visual field. Generally the term hemianopsia is used in the study of brain affections, hemiopia in the description of peripheral disturbances. Hemianopsia is characterized by loss or obscuration of vision in one-half or, more properly speaking, in nearly one-half of the visual field, and the lesion causing this phenomenon may be situated anywhere from the optic chiasm to the cortical centres of vision in the occipital cerebrum. Under "cranial nerves" and "cerebral

localization," the occurrence of lesions causing hemianopsia will be considered. Fig. 143 shows the relations of corresponding halves of the retina to the visual paths and centres in the brain. Instead of hemianopsia it may be necessary to study various partial obscurations of the visual field, as quadrants, sextants, or irregular areas. Hemianopsia may be horizontal, vertical, bitemporal, binasal, or lateral homonymous, the latter being by far the most common. Hemianopsia can be roughly determined by bringing objects into what should be the patient's fields of vision and noting when they are first seen. An instrument known as the perimeter, one form of which is shown in Fig. 144, is used for more careful determinations. It consists of an arc of a circle with an opening in its central portion and carrying two slides, marked in degrees, and so fixed to a standard that it can be rotated and can be placed either vertically or horizontally. The patient, supported by the chin rest, with one eye closed, fixes the other upon the opening, and the slides are brought towards the centre until they come into his field of vision. Charts accompany the instruments.

Fig. 143.

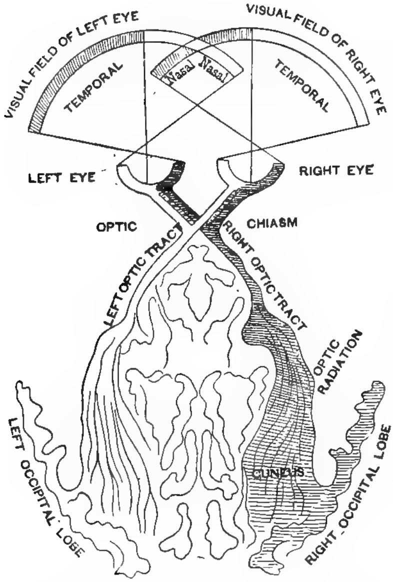
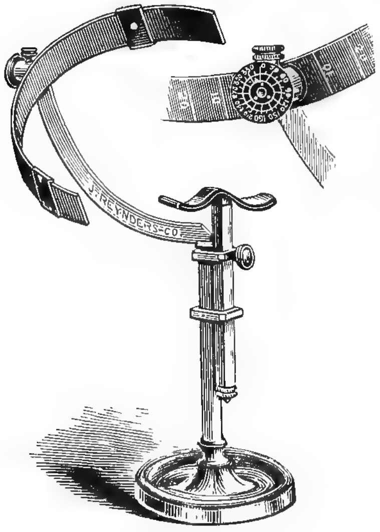


Diagram of the origin, course, and distribution of the nerve fibres of the optic nerve. (Moore.)

Fig. 144.



Perimeter of Emerson.

Wernicke's Hemiopic Pupillary Reaction.—Wernicke was the first to call attention to a peculiar reaction of the pupil in some cases of hemianopsia when a fine pencil of light is thrown on the sensitive half of the retina; contraction of the pupil promptly results, but response is absent on the insensitive half. As the reflex arc between the retina and the iris is through the primary optic centres

in the pregeminum and pregeniculum, if the lesion is behind these centres this arc is intact and the reaction is absent, but if the arc is broken the reaction can be obtained. To test for the hemiopic pupillary reaction, or *inaction*, the patient, being in a dark or nearly dark room, with the lamp or gas lighted behind his head in the usual position, is made to look to the other side of the room, so as to exclude accommodative movements of the iris, and a faint light from a plain mirror, or from a large concave mirror held well out of focus, is thrown upon the eye and the size of the pupil is noted. With the other hand a beam of light focussed from the lamp by an ophthalmoscopic mirror is thrown directly upon the optical centre of the eye, then laterally in various positions, and also from above and below the equator of the eye, the reaction being noted at all angles of incidence of the ray of light. (Seguin.) Instead of a mirror a lens can be used; and the light can be better restricted to one-half of the retina by holding the edge of a card, cut to the shape of the ball, vertically against the open eye.

FIG. 145.

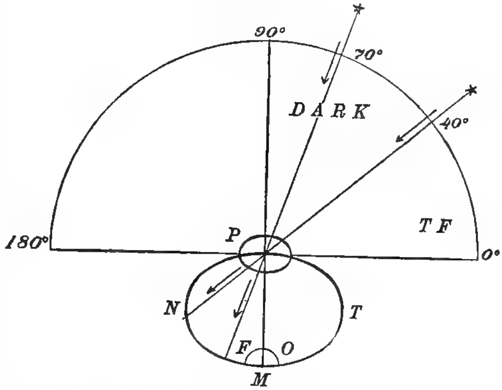


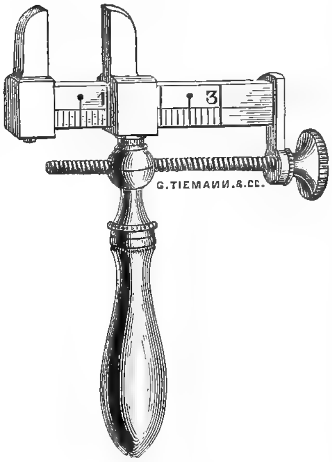
Diagram illustrative of the test for *hemiopic pupillary inaction*; the lines represent a horizontal plane through the left eye and its visual field: F O, fundus oculi; M, macula lutea; N, nasal half of the field which is anesthetic in temporal hemianopsia; T, temporal half of retina; T F, temporal field; P, pupillary aperture; 180° to 0°, the equatorial arc or semicircle; 90°, vertical point and line passing through centre of eye to M; 70° and 40°, rays of light striking the insensitive nasal half of the retina, producing no pupillary reflex. (Seguin.)

Color Changes.—Occasionally color blindness is associated with hemianopsia, or with quadrant or sector defects. Concentric restriction of the field of vision and reversal of the order of the color field may be present, and can be tested with colored disks in the slide of the perimeter. Special abnormalities of the visual field occur in functional diseases of the nervous system and even in health. They may be hysterical, they may be due to exhaustion, or they may be added to the symptoms of organic disease, and yet not be genuine phenomena of such disease. De Schweinitz and J. K. Mitchell have particularly called attention to changes in the visual field in health and in hysteria. *Color blindness* or partial color blindness can be roughly tested by having the patients look at objects of different colors. For careful discriminations the color wools such as are employed in testing railway employees will be found useful. Oliver has recommended the use of loose or separate strands of wool

of pure red, pure green, rose, pure yellow, and pure blue. The colors are made of equal relative intensity, and the value of the color used in each test skein is expressed by a system of metallic bangles made incomprehensible to all but the scientifically initiated. A black surface is used in testing.

Pupillary Symptoms.—Some of the most important pupillary symptoms are differences in the two pupils; contracted or dilated pupils; reflex iridoplegia or impaired response to light (the Argyll-Robertson pupil); abrupt and frequent changes in the pupil; and paralysis or spasm of accommodation. It must be remembered that the pupils are not always equal in the healthy. Moebius found the pupil small among the aged. In soldiers Ivanoff found the two pupils equal in only twenty cases out of one hundred and fifty. In two-thirds of these cases the left pupil and the left half of the face were more developed than the right, and the diameter of the pupil was in direct relationship with the development of the face of the same side and the limbs of the opposite side. Contraction of the pupil, unilateral or bilateral, is present in numerous acute brain affections, as in injuries, meningeal inflammations, and apoplexies. In degenerative organic diseases, as the scleroses, the fixed small pupil, or Argyll-Robertson pupil, is one of the most significant signs. Myelitis may have for one of its manifestations marked pupillary changes. The diameter of the pupil varies between 2.44 and 5.82 mm. In all doubtful cases measurements of the pupil should be made, which can be done simply by holding before it a rule marked in millimetres, or by a pupillometer, such as is shown in Fig. 146. In a pupillometer invented by Follin and modified by Randall, a series of circles held to the observer's eye is revolved until the one which matches the pupil is reached. Other instruments for measuring the pupil are described in works on ophthalmology.

FIG. 146.



Pupillometer.

Mobility of the Iris.—The patient is placed before a window in diffuse daylight, one eye is carefully excluded from the light, and he is directed to look into the distance with the exposed eye, which is then shaded, and, if it is normal, a considerable dilatation of the pupil will occur. On removal of the covering hand or card, contraction to the same size as that which existed before the test takes place. During this examination the other eye will act in unison

with its fellow, and in normal eyes the pupils should be equal, not only with both eyes open, but with one eye shaded. When the covering hand is removed from the eye directed towards the light, the dilatation which existed in the pupil yields to a contraction, succeeded in a moment by a slight dilatation and again a contraction, oscillating thus for a moment until it settles to the original size,—a phenomenon called *hippus*, the explanation of which by Swanzy is that each contraction of the pupil, diminishing the supply of light to the retina, contains in itself the cause of the succeeding dilatation, and, conversely, each dilatation sets in motion the succeeding contraction, until at last equilibrium is attained. During the whole process of testing the reflex mobility of the iris, the observed eye must be steadily fixed upon a distant point; otherwise the influence of accommodation and convergence will arise. (De Schweinitz.)

Hearing.—Having closed the patient's eyes, a watch can be carried from a distance *towards* the ear, and the point at which the tick of the watch is first heard should be noted. The watch should not be carried *from* the patient in making this test. The distance at which the natural tone of the voice is heard by a normal ear should be noted, and using this for comparison the distance at which the patient is able to distinguish spoken words can be tested. The eyes should be closed or blindfolded, as the words spoken might be learned from the motions of the lips. These tests show the extent of "air conduction." Bone conduction can be tested by using the watch and the tuning fork. With the meatus closed by pressure on the antitragus, when the watch is placed at the root of the zygoma or on the mastoid process it is heard in the normal ear better than when the air intervenes between the watch and the tympanic membrane. Diseases and conditions not strictly pathological change this result. In the aged the intensity of sound is diminished on account of physiological degenerative changes. Disease of the labyrinth or of the nerve will diminish or destroy bone hearing. Galton's whistle consists of a piece of brass tubing with an internal diameter of less than one-tenth of an inch, into which a plug is fitted so that it can be drawn out or pushed in at will. For testing tones as to their pitch, whistles of this kind or the tuning fork may be used. With a low pitched tuning fork the lowest vibration that can be perceived as a tone can be determined; the highest tone can be determined with the whistle. Numerous experiments that may be made upon the sense of hearing can be learned from a study of the laboratory courses in physiological psychology.

Gellé's Binaural Reflex.—If pressure or suction is exercised upon the tympanic membrane of one ear by the pneumatic speculum or a Politzer bag closing the canal, the sound of a tuning fork, either on the vertex, on the tube closing the canal, or held free in the air

in front of the opposite ear, will be reduced in loudness if the conducting apparatus of the ear is normal. The pressure is transmitted to the conducting apparatus and reduces the mobility of the stapes in the oval window, while it probably changes the labyrinthine pressure; one or both of which can account for the impaired hearing on the manipulated side. In the opposite ear there seems to be a consensual reflex action of the stapedius muscle, which decreases the mobility of the stapes of that side, with a like resultant reduction of the hearing. Where there is fixation of the conducting apparatus, especially of the stapes, no effect is produced by such pressure or suction. When the vibrating tuning fork is placed before one ear, while pressure is made by a Politzer bag fitted to the opposite canal, the patient perceives the diminution of the sound of the fork, due to the synergetic bilateral tension of the auditory apparatus; this is the test of the binaural reflexes; and this exploratory procedure makes manifest the mobility of the stapes, the activity of its motors, and their variations. Clinical experience shows that disappearance of the binaural reflex especially coincides with lesions of the cervical cord. The centre of the reflex movements of binaural accommodation is situated in the cervical cord. The test is of value when disease of the ear is not present. This reflex does not appear to be related to the pupillary reflex; either may persist when the other has been lost.

FIG. 147.

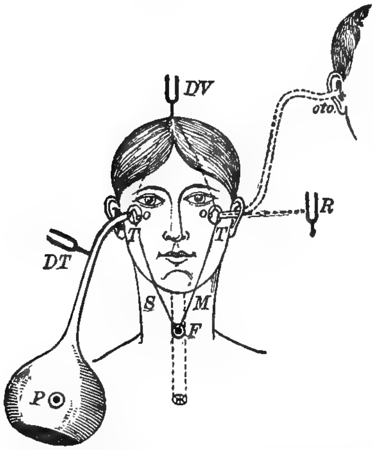


Diagram illustrating the binaural reflex: DV, the tuning fork at the vertex, the dotted lines going from which lead to the stapes and indicate the road followed by the cranial sonorous waves; DT, the tuning fork placed on the tube which unites the air bulb to the ear, the sound following the tube to the tympanic membrane; P, the air bulb, on compressing which immediately the sound of DT is diminished; in the same way the sound of DV diminishes through the action of the stapes of the same side; *oto*, otoscope, with which the observer auscults by the other ear the sound of DV at the moment of pressure, and perceives the attenuation of sound; R, the tuning fork vibrating in front of the ear opposite to that which is submitted to pressure; the subject perceives the sound equally weakened; S, imaginary line which extends from the sonorous conducting apparatus to the cervical spinal cord, and conducts the sensation of passive movement due to pressure to the reflex nervous centre (F); M, imaginary line connecting the cervical spinal cord to the transmitting apparatus of the opposite ear, by which passes the synergetic motor excitation of binaural accommodation. (Gellé.)

Smell.—Among the substances which may be selected for testing the sense of smell are musk, camphor, oil of cloves, yellow wax, rose water, valerian, and asafetida. Solutions are made of different strengths, and for fine determination, that is, of minimal odors, the patient can be required to smell in succession of the bottles, which are kept at some distance from each other, indicating

when he first detects an odor. Pungent substances, such as ammonia, should not be used.

Taste.—Standard solutions, either clear or mucilaginous, of sweet, sour, salt, and bitter substances should be made, as of sugar, citric acid, salt, and quinine. Sanford gives the following as convenient. Stronger solutions: sugar, 40 : 100; quinine, 2 : 100; tartaric acid, 5 : 100; salt, saturated solution. Weaker solutions: sugar, 5 : 100; quinine, 2 : 100,000; tartaric acid, 5 : 1000; salt, 2 : 100. The solutions, however, need not be so carefully prepared for ordinary clinical work. The patient puts out the tongue and is instructed to keep it out, and the solution is put upon the tongue with a camel's-hair brush, rinsing the mouth as often as necessary. It should be applied to the tip, sides, back, and middle of the tongue according to the part desired to be tested, and to each half carefully. The words "sweet," "sour," "bitter," "salt," and "cannot tell" having been written on a slip of paper, he is directed to answer by pointing to the proper expressions. This sense can be electrically examined by an instrument invented by Neumann, which consists of a long stem carrying two wires isolated from each other and ending in little balls, which form two poles of the current. By means of this electrode any part of the tongue or inside of the buccal cavity may be examined and the sensations of taste noted. Instead of this, one electrode of a galvanic battery may be placed above and another under the tongue, or one on the tongue and the other on the back part of the neck. Much of what is termed taste is really smell, or touch, or both; to prove which, with eyes shut and nostrils held, try to distinguish by taste alone scraped apple and scraped potato placed in succession on the tongue.

Motor Disorders.—Motor disturbances include such phenomena as paralysis, complete or partial; spasm, local, unilateral, or general; contractures and local spastic states; tremor, massive or fibrillary; and incoordination. Electrical conditions of the nerve and muscle belong in part under disorders of motility, but will be considered as a special section. Paralysis may be partial or complete, and may assume numerous forms, as *hemiplegia*, where it attacks the most of one half of the body; *monoplegia*, where it is shown in one limb; *paraplegia*, where it affects both lower extremities; and *diplegia*, where it affects corresponding extremities, as the two legs, or the two arms, or all four limbs. A movement made with the unparalyzed limb, or, more rarely, when some power is preserved, by the paralyzed member, can cause movement on the other side. These are called *associated movements*. A *forced movement* is one in which the tendency is to move in spite of the patient's will in a certain direction—to one side, or forward, or backward—or to rotate. Movement may be abnormally increased,

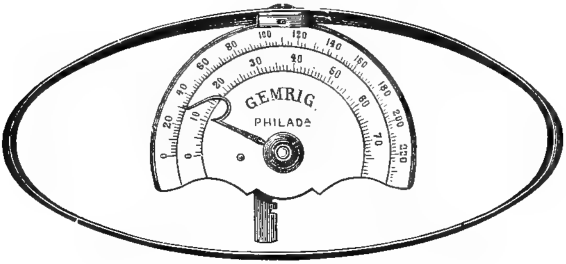
giving rise to *spasm*, *convulsion*, or *tremor*. Marie, Charcot, Grashey, Huber, Gowers, and Peterson have studied tremor with the assistance of the sphygmograph, in paralysis agitans, exophthalmic goitre, general paresis, sclerosis, and other affections. Peterson, using the Edwards sphygmograph, obtained beautiful transfers of muscular movements on paper. The sphygmograph is firmly fastened to a table, and the finger or part to be tested is placed against the lever, the will of the patient being as far as possible excluded. Contracture is muscular shortening not due to structural changes in the muscle, although the condition is frequently dependent upon organic disease involving the motor centres or tracts in the brain or spinal cord. Contractures are also present in grave hysteria, and lasting for years may lead to the diagnosis of organic disease. As distinguished from contracture, the word *contraction* is sometimes used to describe muscle shortening accompanied by structural changes. Spasm or convulsion may be local, unilateral, or general; it may be *clonic*, when the spasmodic movements are rapidly intermittent, or *tonic*, when the contraction persists for a definite period. Sometimes tonic spasm is so persistent and severe as to cause *rigidity* in the extremities, neck, or trunk. *Conjugate deviation of the eyes and head* is a motor phenomenon often present in diseases of one side of the brain; the eyes are directed to one side, sometimes with and sometimes without a corresponding rotation of the head.

Examinations for Paralysis.—In hemiplegia an examination should be made systematically from above downward, or the reverse. The patient should be directed to rotate the head first in one direction and then in the other, to bend it forward or backward, or to perform any special movement. The ocular movements should next be studied, and the pupillary movements as described under reflexes. If the case is one of monoplegia or any form of local paralysis, of course only the parts affected need be examined and compared. For the upper extremities movements of elevation, projection, retraction, supination, flexion and extension, and combination movements, should be studied. Every isolated movement and every grouping of movements should receive attention in succession, from the simple flexion of the index or little finger and the coarser movements of the elbow, upper arm, and shoulder, to the most complicated movements of the forearm and hand. Both cerebral and spinal paralyses have a tendency to arrange themselves in upper, middle, and lower arm types, according to the degree in which different parts of the limb are affected. The movements of the leg can be studied with more facility, but some regular system should be observed. Movements below the knee and some of those above the knee are best studied in an easy sitting posture. The patient thus placed should be directed to bend the foot upward on the

heel, or on the toes, to move it inward or outward, and to perform the various intermediate movements; he should be made to try to project the leg forward and backward, to semirotate it, to move the thigh upon the pelvis, to abduct and adduct the thighs, to throw one leg over the other, and to place the foot over the opposite thigh. Standing on the sound limb if hemiplegic, and if necessary supporting himself, he should try to move the limb in various directions from the hip joint. The few trunk movements are easily tested after the same methods.

The Mathieu Dynamometer.—The strength or motor power of patients is sometimes tested by means of the *dynamometer*, or measurer of power. The most common form of this instrument, invented by Mathieu (Fig. 148), is an elliptical steel spring attached to a semicircular scale, around which an indicator is free to move. This is connected by cogwork with a steel arm, the lower end of

Fig. 148.



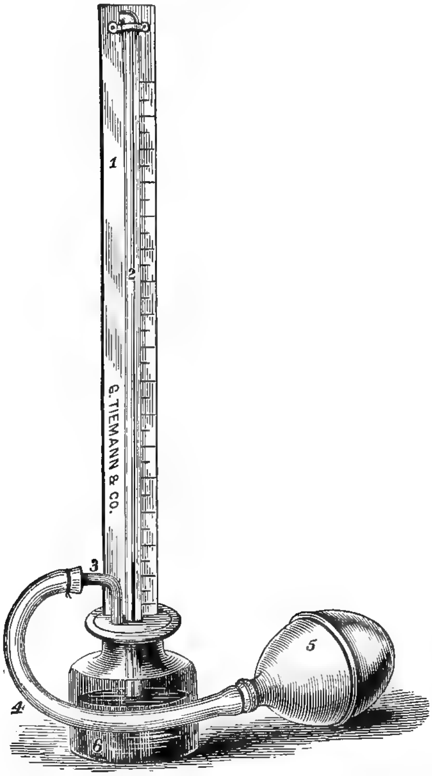
The Mathieu dynamometer.

which touches the spring where the index points to zero, one end of the scale being marked zero, the other 100. The 100 degrees represent the distance to which the grip of a vigorous adult can drive the indicator. To use the instrument it is grasped as firmly as possible by the hand, and the two sides of the spring are pressed together steadily. The left hand can be compared with the right, or the grip of a sick man with that of one who is healthy. A dynamograph for graphically representing the muscular strength can be made by attaching to the dynamometer a toggle joint which moves a steel rod carrying a pencil.

The Hamilton Dynamometer.—McLane Hamilton, recognizing the disadvantages of the Mathieu dynamometer, devised another form (Fig. 149). For recording results he combined the rubber bulb with the drum of Marey. The drum has two pipes, one of which is connected with the rubber bulb, while another is attached to the lower end of an open glass tube, these being partly filled with colored fluid. The fluid is forced upward by the pressure of the patient's hand. The sustained voluntary effort required to keep the fluid at

this point necessitates some delicacy of muscular coordination, and should this be impaired there will be expansion of the drum head and consequently irregular tracings upon the cylinder of the registering apparatus, which should be covered by a piece of smoked paper, and the stylet placed in apposition thereto.

FIG. 149.

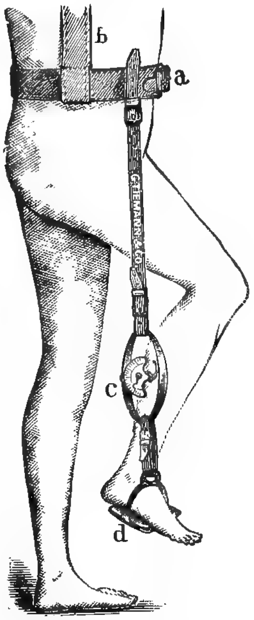


Hamilton's dynamometer: 1, scale marked on one side in pounds and on the other marked at regular intervals for making comparative estimates; 2, glass tube with closed end; 3, bent brass tube (connected by a rubber tube, 4, with a closed rubber bulb, 5); 6, bottle filled with mercury.

Pedodynamometers.—Pedodynamometers or foot dynamometers of various forms have been invented by Birdsall, Dana, Féré, Krauss, and others. Most of these instruments are so made as to measure strength by the pressure of the foot or the pull of the leg. All have some disadvantages. One of the best is that invented by Krauss, and shown in Fig. 150. The harness consists of a wide heavy belt (*a*), its inner surface padded so that its adjustment around the waist will not be uncomfortable. A heavy webbing (*b*) is looped through the belt, passing over the shoulders, and helps to retain the belt in proper position. A dynamometer (*c*) is connected with the belt by means of a strong, adjustable strap, permitting it to be lengthened or shortened according to the stature of the patient. Connected with the dynamometer is a stirrup (*d*), the base of which is also padded for receiving the foot. Pressure exerted upon the stirrup will be registered upon the dial of the dynamometer, and the exact strength of the extensors of the leg can be ascertained. The apparatus can be applied as shown in the illustration, viz., the patient in a standing position, or he may take the recumbent position. The leg should be flexed, making an angle of 135° at the hip joint and an angle of 90° at the knee joint. By lengthening the strap which connects the dynamometer (*c*) with the belt (*a*), and flexing the foot on the leg as much as possible, then directing the patient to extend the foot forcibly, the power of the calf muscles can also be determined.

By using snaps, the dynamometer can be quickly removed and used to measure the power of the hands.

FIG. 150.



Krauss's pedodynamometer.

Cutaneous Reflexes.—Certain points in symptomatology are determined by a study of the normal, of the undue, or of the lacking response on stimulating the skin. Undue response, for example, may indicate that spinal centres are in an irritable or inflamed state, or that they are cut off from central control, or that cerebral centres are diseased; lack of response, that the nerve tracts in the periphery or in the cord are injured or that the spinal centres are impaired or destroyed. These cutaneous reflexes have for convenience been considerably subdivided, according to the areas of the body chosen as most convenient or desirable for making the tests, as into the *plantar*, obtained from the sole of the foot, the *abdominal*, from the sides of the abdomen, the *epigastric*, from the epigastric region, the *cremasteric*, from the inner part of the thigh; and in the trunk, upper extremities, and head, the *intercostal*, *interscapular*, *palmar*, *bicipital*, *tricipital*, *conjunctival*, and *pharyngeal*.

Frequency of the Occurrence of Reflexes.—Under Geigel's direction, Plaesterer investigated the frequency with which the reflexes occurred in one hundred men and one hundred women not the subjects of nervous disease, with the following results:

	100 men.	100 women.
The epigastric	62 per cent.	
The abdominal	99 "	92 per cent.
The intercostal		16 "
The cremasteric	66 "	
The plantar	98 "	88 "
The interscapular	15 "	13 "
The gluteal	28 "	11 "
The tibial periosteal	5 "	
The periosteal at wrist	29 "	
The patellar	98 "	

The skin reflexes are easily exhausted. Even in very sensitive persons, who at first squirm on an attempt to obtain the plantar and other reflexes, later attempts may be fruitless. Plaesterer therefore suggests that in examining the skin reflexes the first irritation be decided, so as to obtain the reflex at once, if present, as later attempts are very likely to be misleading.

Ataxia.—By *ataxia* is meant incoordination or inharmonious muscular action. It may be static or dynamic. It is due to disease or functional disturbance of some part of the peripheral or central apparatus concerned in maintaining equilibrium. In multiple neuritis, and in ataxias after infectious diseases, the peripheral nerves are chiefly at fault. When it is a symptom in brain tumor, the lesion is commonly located in the central lobe of the cerebellum; in locomotor ataxia the degenerative lesion has attacked the posterior spinal ganglia, nerve roots, and columns of the spinal cord. It is through lesions thus situated that the sensibility of skin, muscles, tendons, and joints is impaired, and the ataxia results largely because of the interference with the ability to discriminate as to movements, pressure, weight, and posture. Ataxia when well marked is readily determined, and is exhibited by the swaying of the body when the eyes are closed; by unsteadiness on one foot with the eyes closed or open; by crossing one leg over the other when standing or sitting; by the gait, and by the method when walking of beginning, halting, or turning around. Ataxia in the upper extremities can be studied by observing the patient separating his hands and arms and then trying to bring the points of his fingers together; by having him touch the ear, eye, or nose, or some other object, with one finger; by his manner of writing or drawing, of buttoning or unbuttoning the clothes, and of picking up small objects. All studies of the so-called muscular sense here come into play.

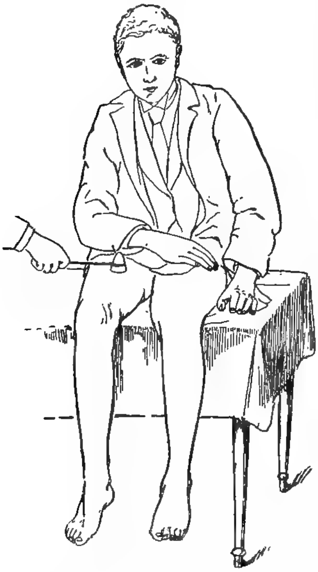
Station and Sway.—Station is the ability to preserve an erect and steady position while standing; and its study is of course of particular value in the investigation of ataxic disorders like tabes. The Romberg or Brauch-Romberg sign has long been known. It is the swaying of the body when the eyes are closed and the feet are brought near together. In extreme cases it may be present when the eyes are open. It is necessary first to have a clear idea as to *normal sway*, instruments for studying which have been invented by Mitchell, by Lewis, and by Hinsdale and Reichert. Careful observations have shown that the normal man and woman have about half an inch sway forward and backward, and three-quarters of an inch laterally; that children sway to a greater extent than adults, and that both in children and in adults closing the eyes increases the sway about fifty per cent. To observe the sway of a healthy man, the subject is made to stand with his heels and toes together, his hands allowed to hang by his sides; the head is held erect, the eyes are directed to a fixed point. A record of motions in the anteroposterior line can be made by attaching a silk thread to a fillet over the forehead; the thread is passed over a pulley and is connected with a rod moving vertically and carrying an index. This index marks on smoked paper attached to a drum which revolves laterally by means of clockwork. Other tracings

can be taken by attaching to the top of a man's head a flat piece of card-board, upon which is stretched some smoked paper. (Hinsdale.)

Deep Reflexes, or Muscle and Tendon Phenomena.—Important signs and symptoms are obtained by mechanical excitations of tendons and muscles, which give rise to a variety of jerks and other movements, the most important of which are the *knee jerk* or *patellar reflex*, *muscle jerk*, *ankle clonus* or *foot phenomenon*, *toe jerk*, *biceps*, *triceps*, and *extensor jerks* in the upper extremities, and the *chin* or *jaw jerk*. Related phenomena are the *paradoxical contraction* of Westphal and the numerous movements observed from tapping bones, as the tibia and the ulna. Knee jerk will be considered in a special paragraph. Ankle clonus or foot clonus, called also the ankle or foot phenomenon, is elicited by rapidly putting the foot into dorsal flexion. The patient sitting as in the study of the knee jerk, with one leg loosely thrown over the other, or, still better, upon a high table or a bed, so that the limb will hang freely, holds the limb half extended. The hand then presses the foot upward; the tendo Achillis is made tense, and the foot is thrown into tremor or vibration, which usually stops when the pressure is withdrawn, and can also be halted by forcibly bending the foot downward. Sinkler has described the *toe reflex*, which is met with only when knee jerk and ankle clonus and the other tendon reflexes are strongly developed. The observer grasps and flexes strongly the great toe of the patient, who lies on his back with legs extended; immediately involuntary flexion of the foot follows, then flexion of the leg, and finally the thigh is flexed on the pelvis. Westphal's paradoxical contraction is a tetanic contraction of a muscle after it has been suddenly relaxed, which is rarely observed in other muscles than the tibialis anticus. The foot is flexed on the leg, when the muscle for some time remains in a state of contraction, and only gradually relaxes and allows the foot to return to the normal position of rest. *Elbow jerks* can be elicited by striking the tendons of either the triceps or the biceps; *wrist jerk* by tapping either the flexors or the extensors of the forearm, particularly the extensors; in fact, response may be obtained from almost any of the prominent muscles of the arm or forearm. Most of these are present in health, but they may be exaggerated, depressed, or abolished in disease of the nervous system. In testing, the limb should be well supported, voluntary efforts on the part of the patient being excluded. Jerks or reflexes can also be obtained from some of the muscles and tendons of the neck. Morris Lewis has described a *chin jerk* or *jaw jerk*. With the mouth open the lower jaw is supported in the hand of the examiner, who strikes with his fingers or a light hammer the teeth or some part of the lower jaw; and if the phenomenon can be evoked, the jaw is at once quickly lifted. The *muscle jerk* is obtained by

striking the muscle, when a certain length of fibre shortens. If disease or injury severs the motor or sensory nerves of a muscle or damages its related spinal centre, the tendon jerk ceases, but the muscle jerk from a direct blow remains. Muscle jerk may be increased by the same disease that destroys knee jerk; it is reinforcible by distant motion or by sensation. Fig. 151 shows the method of taking the muscle jerk by striking the quadriceps femoris.

Fig. 151.



Method of taking muscle jerk.

Knee Jerk or Patellar Phenomenon.—When the limb of a normal individual is semiflexed and supported and the knee pan is gently struck, the foot and leg are projected forward, the amount of the movement differing considerably even in health, but very rarely being absent. The variation of the knee jerk in health or in mere functional disturbances should first be understood. It may be diminished in old age; it may be sluggish in certain conditions of the system; in hysterical and neurasthenic states it is likely to be exaggerated; it may vary in the same person even in the same day; it is much influenced particularly by emotions and by mental and physical exhaustion. Marked differences between the two sides of the body are often valuable in diagnosis, as the normal condition is one of bilateral uniformity. Eulenberg found that of seven children who were examined during the first twenty-four hours after birth, the knee phenomenon was very distinct in six. In one boy, thirteen days old, who was suffering from atrophy, it was wanting, but it was present in all the other children between one and four years who were examined. As a rule, it was more marked on one side. It was wanting only in seven with imperfect nutrition, out of one hundred and seventy-three infants over one month old; and in children from two to ten years of age its absence was as exceptional.

Method of Taking the Knee Jerk.—In studying the knee jerk the limb should be supported so as to remove from it the influence of the patient's will. One leg as much relaxed as possible is thrown over the other, or the physician supports the leg with his hand or arm, or the patient sits upon a bed or table or high stool so that the limbs will dangle freely. The blow can be given by the ulnar border of the hand, by the palmar tip of the index or middle finger, or by a percussion hammer. (Fig. 152.) The sign can be

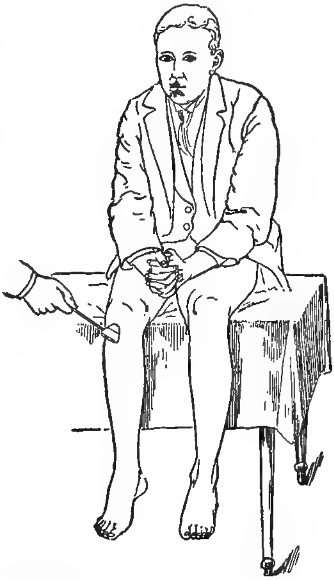
elicited through the clothing, but in doubtful cases it is better to have the skin uncovered. To study this phenomenon with the patient in bed, it is best to have the leg supported at the ankle or foot as well as at the knee, and even then it is sometimes diffi-

FIG. 152.

Taylor's percussion hammer.

cult to get complete relaxation. By putting the finger of one hand across the quadriceps tendon just above the patella, pushing the latter downward, and percussing the finger suddenly, not only the knee jerk but sometimes clonus can be elicited. (Gowers.) An active willed movement made by the patient at the time of percussion of the patella increases the knee jerk. This is called *reinforcement*, and has been especially studied by Jendrássik, Weir Mitchell, Lewis, Lombard, and Bowditch. Usually it is practised by having the patient during the time of examination constantly engaged in pulling upon his interclasped fingers or hand (Fig. 153). By this procedure a knee jerk which is absent can be brought out, and when sluggish or diminished it may be much increased. In recording examinations, symbols or abbreviations may be used, as *k. j.* for knee jerk; *m. j.* for muscle jerk; *rf.* for reinforcement; + for increase; — for decrease. Weir Mitchell has invented a simple apparatus to get a numerical statement of the knee jerk for reference. A yardstick carries a light movable arrangement, about four inches in height by five inches in breadth, which is so set that the toe rising with the jerk pushes it up the scale. As it moves, it pushes up and leaves for a marker a double loop of wire. Lombard has invented a more elaborate apparatus, but it is not well adapted to clinical work except in hospital or office neurological practice.

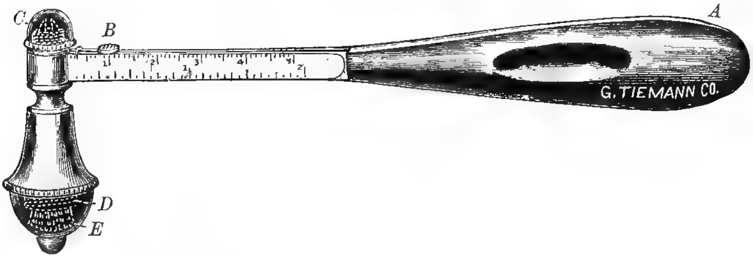
FIG. 153.



Method of taking knee jerk with reinforcement.

Krauss's Combination Percussion Hammer and Esthesiometer.—This hammer has a heavy metallic head fixed to a flattened oval handle. As a hammer it may be used to examine the tendon and muscular phenomena, to percuss the head, spine, superficial nerves, etc. The handle (*A*) being of hard rubber becomes *warm* on friction, while the head being of metal is *cold*, thus offering a means of examining the sense of heat and cold. The cap *C* when

FIG. 154.



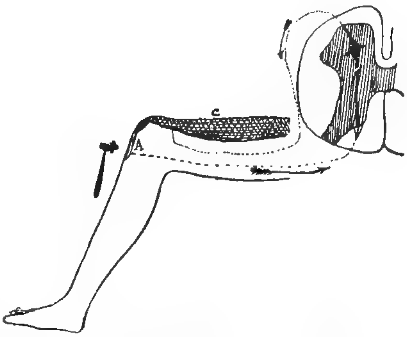
Krauss's combination hammer and esthesiometer.

removed discloses a triangular spear head one-half centimetre long, while at the other end of the hammer head is the blunt rubber point, giving *sharp* and *dull* points for examining for anesthesia and hyperesthesia. The spear is divisible into two portions, one fixed upon the hammer head, the other movable upon a metallic slide (*B*) forming part of the handle (*A*), upon which are engraved the English and French scales. This arrangement furnishes an excellent *esthesiometer*. Replacing the cap *C* and removing the cap *E*, a camel's-hair brush is exposed, furnishing a *soft* surface, while the metallic cap *C* presents a *hard* surface.

Physiology of the Knee Jerk.—C. Sachs demonstrated the existence of nerves in the tendons. Westphal believed that the muscular contractions were not reflex manifestations, but were produced by direct irritation of the muscular substance; Joffroy held that the phenomena were usually due to irritation of the skin. Destruction of the spinal roots in the middle and lower lumbar region causes the knee jerk to cease. The crural nerve in the rabbit takes its origin between these vertebræ. According to Sherrington, the knee jerk has an intimate dependence upon the integrity of the reflex arc. He found that the whole of the quadriceps extensor or of the anterior crural nerve is not necessary for the "jerks," but that they depend upon the vastus internus muscle, and perhaps the subcrureus and branches of the anterior crural nerve. Fig. 155 is a scheme, after Marie, of the patellar reflex or knee jerk. The tendon (A) of the patella (B) having been struck, the excitation is carried to the spinal cord by crural sensory fibres, and enters by the dorsal roots into the dorsal column, and thence passes to the dorsal horn; next it

takes its course through the gray substance until it reaches the cells of the ventral horn, and thence the motor excitation goes through the motor root and nerve to the muscle (C). Although Lombard and others believe the knee jerk is not a pure reflex, absence of this phenomenon follows a break in any portion of this path. Lombard and de Watteville hold correctly that the jerk is due first to direct stimulation of muscle, and secondly to reflex influence.

Fig. 155.



Scheme of the patellar reflex path. (Marie.)

Cremasteric Reflex.—The cremasteric reflex, which is elicited by irritating the inner surface of the thighs and thereby causing retraction of the testicle and scrotum, has been studied by Mitchell, Rosenbach, Jastrowsky, Hinze, and others, and is of considerable importance in some toxic and degenerative diseases of the spinal cord. This reflex differs in youth, adult life, and old age. In the healthy boy it may cover the inner third of the thigh, ceasing near the middle line in front, and it may even extend to the calf; in the adult it is more restricted and usually ends above the knee; its area and intensity decrease in old age. Recently I have developed it by irritation of the skin as low as the ankle in a case of myeloneuritis. Usually excitation on one side will produce the reflex only on the same side; but in conditions of disease, and probably also occasionally in health, the contraction occurs on both sides; sometimes in disease the reflex is crossed. The cremaster is a portion of the lower edge of the internal oblique and transversalis muscles. The reflex arc arises in the skin by nerve twigs of the anterior crural, internal cutaneous, middle cutaneous, two saphenous, and obturator; and it is completed through the lumbar cord by the genital branch of the genitocrural trunk. Geigel endeavored to ascertain whether a reflex equivalent to the cremasteric in the male existed in the female. He found that irritation of the inside of the thigh was followed by a contraction of the lowest fibres of the internal oblique muscle in ninety-six females. It was absent four times. In three female hemiplegics it was missing on the paralyzed side. He proposes to substitute the name "oblique or inguinal reflex" for cremasteric reflex.

Bulbocavernous Reflex.—Under this name Onanoff describes a sudden contraction of the ischial and bulbocavernous muscles caused by mechanical excitation of the glans penis. It is elicited by placing the left index finger on the bulbous portion of the penis, and with

the right hand stroking with a piece of paper the dorsal surface of the glans, or pinching lightly the mucous membrane. Onanoff believes that the reflex is present in normal man, and that its absence may be the sign of an organic lesion. If the sexual function is impaired and the reflex is present, the affection is probably of dynamic origin and a favorable prognosis can be given. Under the name of *virile reflex* Hughes has described a like phenomenon.

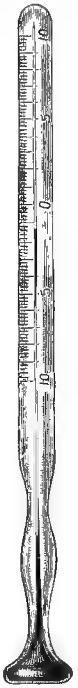
Anal Reflex.—Rossolimo has described an *anal reflex*, and has concluded from experiments upon dogs that its centre is the segment of the cord which corresponds to the third sacral root. He found the reflex augmented in neurasthenia and in myelitis located high in the cord, but diminished in sacral neuritis and in posterior sclerosis.

Vasomotor, Trophic, and Secretory Symptoms.—Vasomotor symptoms, such as pallor or flushing, erythema, heat or cold, goose skin, lividity, ischemia, and edema, are studied by inspection, by careful investigation of surface temperature, and, when swelling is present, by measurements of the parts. Vasomotor affections may be spasmodic, paralytic, or even ataxic. Trophic or nutritive affections, sometimes called trophoneuroses, chiefly affect the skin, muscles, bones, and joints, and include atrophies and hypertrophies; perforating ulcers of the limbs; corneal ulceration; gangrenous patches, usually symmetrical; cutaneous eruptions, as herpes zoster, papules, and pustules; acute bedsores; bone arrests or overgrowths and abnormal brittleness of bones; swellings and effusions in joints, called arthropathies; overgrowth or undergrowth of hair, or changes in its color; deformed, furrowed, curved, or discolored nails. The methods of studying such disturbances are of course included under various heads, as inspection, measurement, and temperature. Various disturbances of the secretory and excretory organs of the body may be due to nervous disease, and their methods of study are largely included under other subjects. Local or unilateral sweating or profuse general sweating, or defect or absence of this secretion, may claim attention, as may also bloody sweating, known as *hemidrosis*, and *chromidrosis* or black sweat, said to be due to the presence of indican. These disorders are to be studied by chemical analysis of the secretions, and by an investigation of the skin and general system. Wood has described, under the name of *ataxic lymphopathy*, a sudden swelling of the lymphatic glands which sometimes accompanies the pain crises of locomotor ataxia. It is only necessary briefly to call attention to the importance of examinations of the urine, not only for albumen, sugar, casts, and specific gravity, but also for uric and oxalic acid.

Surface Thermometry.—The surface thermometer is an instrument for determining local temperature, and can be used with advantage in infantile paralysis, in some local palsies, and in various

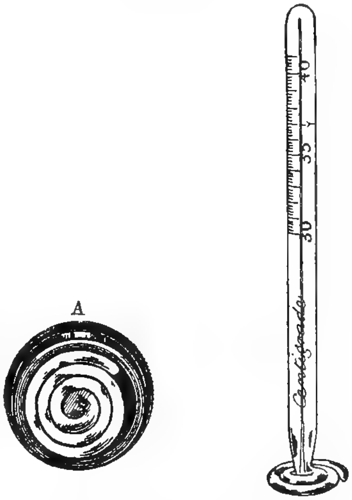
vasomotor and trophic affections. In cerebral disorders it may show a high temperature on one side or on one part of the head. We owe it chiefly to the elder Seguin. In the original Seguin instrument (Fig. 156) the bulb was made flat so as to rest readily, and was carefully graduated; but it was too sensitive to pressure and variations of the air to be of much service. In Mattson's thermometer the bulb is a flat spiral which will not yield much to pressure. It is usually enclosed in a circular box of hard rubber. The spiral and rim of this box are shown at A in Fig. 157. Broca and L. C. Gray have recommended certain stations on the head, shown in Fig. 158, as the most suitable for making surface thermometrical observations. They are (1) the *frontal stations*, one on each side somewhat back of and above the commencement of the external angular process of the frontal bone; (2) the *parietal stations*, one on each side just above the ear; and (3) the *vertical stations*, over the fissure of Rolando, two on each side. The thermometers can be held in place by a strip of perforated soft morocco running around and over the head. Observations in surface thermometry are made by comparison of diseased with healthy parts. Generally two or more surface thermometers are employed at the same time. Having two exactly alike, they are applied perpendicularly and without marked pressure to the skin. They can be left three or four minutes *in situ* and read, and then two or three minutes more. The mouth, rectal, or axillary temperature may at the same time be determined. Instead of the mercurial surface thermometers of Seguin and others, the thermoelectric pile may be adapted to the purposes of local thermometry. Lombard made many thousand observations on the regional temperature of the head, using chiefly a thermoelectric apparatus; and Lombard and Haynes, and also Amidon, studied the effects of willed muscular movements on head temperatures, the latter using in his experiments a modification of Seguin's surface thermometer. Variations in normal surface temperatures both absolute and relative must be fully taken into account.

FIG. 156.



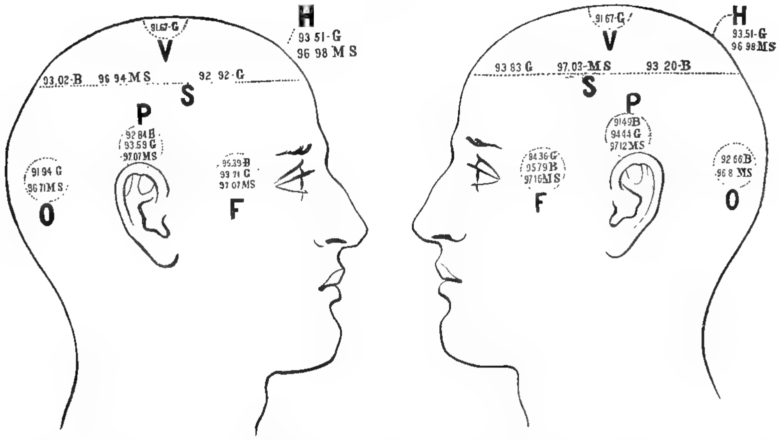
Seguin's surface thermometer.

FIG. 157.



Mattson's surface thermometer.

FIG. 158.



Diagrams of normal cranial temperatures: B, figures of Broca; G, figures of Gray; MS, figures of Maragliano and Seppilli; F, frontal station; P, parietal station; O, occipital station; V, vertex station; H, entire head; S, whole side of head. The observations of Maragliano and Seppilli were made in summer, and those of Broca and Gray in cold or cool weather, which is the reason of the discrepancy in the averages.

The Pulse and Arterial Pressure.—In monoplegias and hemiplegias, and in various lesions of the nervous system due to vascular disease, conclusions as to causation and treatment may need to be based more upon the study of the heart, of the pulse, and of arterial pressure than upon a consideration of the actual textural injuries suffered by the nervous system. Abnormally high or abnormally low arterial tension, compressibility or incompressibility of the pulse, and the variations of pulse associated with various forms of valvular disease in cerebral embolism and hemorrhage, may need consideration. Sphygmographic studies of the pulse occasionally yield interesting results. Féré has made such studies in hemiplegia, finding pressure constantly diminished on the affected side. The same has been observed in hysterical hemiplegia. A study of this kind may be a means of detecting simulation. A comparison of the pulses of the two sides of the body by the ordinary manual methods will sometimes reveal interesting differences. Inequality of volume is not infrequent. Brain disease by affecting the cardiac centres in the oblongata may cause either slowness, increased frequency, or irregularity of the pulse. Arteriocapillary sclerosis plays an important part in the etiology of many lesions occurring in the nervous system. Headache, vertigo, insomnia, want of concentrating power, gradual mental failure, and general weakness are frequently due to arteriocapillary sclerosis, but are supposed by patients or physicians to be evidences of tumor or meningitis, or perhaps only of neurasthenia.

ELECTROPHYSICS AND ELECTROMEDICAL APPARATUS.

Galvanism, Faradism, and Franklinism.—*Galvanism* as used in medicine always refers to the current obtained directly from a cell or a series of cells; *faradism*, to that derived from an induction coil. Synonyms for galvanism are *voltaic*, *dynamic*, *chemical*, or *contact* electricity, or the *constant*, *continuous*, or *direct* current; and for faradism *induction electricity*, *electromagnetism*, the *coil current*, and the *interrupted current*. *Franklinism* is the electricity obtained from a static machine. Galvanism, faradism, and franklinism are not essentially different in their nature. A current can be generated by magnetic induction which will produce all the effects of galvanism and franklinism; with a galvanic battery and suitable changing devices, effects identical with those regarded as peculiar to faradism and franklinism can be obtained; and it is also possible with suitable electrostatic generators to produce the effects of galvanism and faradism. With all three it is simply a question of volts, ohms, and amperes with certain special phenomena.

Action of Electricity on Living Tissues.—The electric current contracts protoplasm, and its effects can sometimes be limited to special tissues. It may act by *electrolysis*, or chemical decomposition of the tissues at the electrodes, and in the circuit; or by *cataphoresis*,—that is, by carrying particles to and through the tissues in the direction of the galvanic current. A modification of nerve excitability known as *electrotonus* occurs in the immediate vicinity of the electrodes or poles and in the interpolar region. At and near the anode the excitability is diminished, producing *anelectrotonus;* while at and near the cathode it is increased, the condition being termed *catelectrotonus*. When catelectrotonus appears by making the circuit with the cathode the nerve is excited by an active modification; when anelectrotonus disappears by breaking at the anode, the diminished excitability is substituted by a negative increase, so that by this procedure also the nerve is thrown into a state of excitement.

Electrophysics.—The *potential* of a substance is its power of overcoming resistance. Separation of electricities takes place at the point of contact of the elements of a cell; the electricity is said to flow from one of higher to one of lower potential, as water flows from a higher to a lower level, or as heat passes from a hot body to one that is cooler. The earth is supposed to be at zero potential, and when a body is at low potential, electricity flows to it from the earth, and the reverse. *Electromotive force* is an imaginary force which causes the difference in potential. *Resistance* is the obstacle offered to the passage of an electrical *current*, which latter is simply the action which continues in the electrical circuit as long as the difference of potential is maintained. The current strength is equal

to the electromotive force divided by the resistance. A *volt* is the unit of electromotive force, an *ohm* the unit of resistance, and an *ampere* the unit of current strength. These terms have concrete interpretations. An ohm, for example, is the resistance to a current of electricity offered by a column of mercury one square millimetre in area of cross section and 106 centimetres long at a temperature of 0° C. or 32° F. The electromotive force necessary to overcome this resistance is estimated to be a little less than that of a freshly charged Daniell's cell. The ampere is the current resulting from the action of one volt on one ohm. The term *milliampere*, introduced by de Watteville, is applied to the resultant of the resistance offered by a human body to a current generated by three Daniell's elements. It is the one-thousandth of an ampere. The *density* of a current is the amount of electrical charge accumulated at any point, and is proportional to the unit area at that point. When a current flows between a large and a small conductor the electricity is denser at the latter, a fact which underlies the scientific use of large and small electrodes. Density is greater at projecting points or edges. A current is ten times denser in a wire one millimetre in diameter than in one of a diameter of one centimetre. If two small wet electrodes are firmly applied over a nerve, the two

FIG. 159.

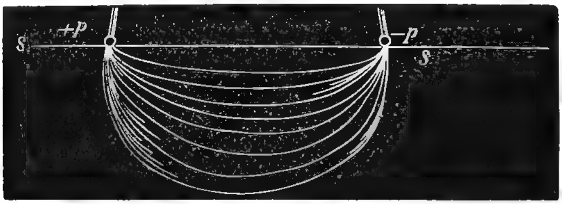


Diagram showing course of currents when two small electrodes are used: $+p$ and $-p$, wetted electrodes; s, skin.

FIG. 160.

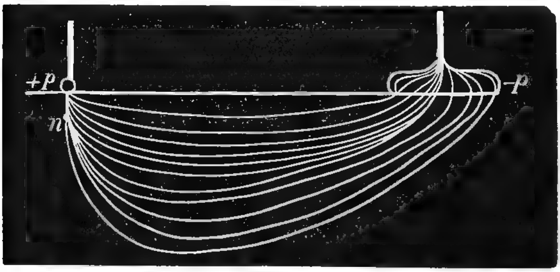


Diagram showing course of currents when one small and one large electrode are used: n, nerve.

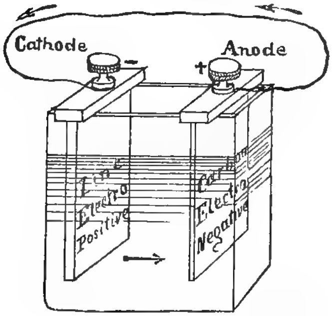
points will receive an equal density of current (Fig. 159); but if one is large and the other small (Fig. 160), the spot under the small electrode will receive the full force of the current, which is diffused in the tissue and over the surface of the other and much larger elec-

trode. A substance along which electricity passes readily is known as a *conductor*; relatively to other substances it presents a low resistance. An *insulator* or *nonconductor* is a substance which does not allow electricity to pass freely, relatively presenting a high resistance. Important conductors are the metals, carbon, flame, linen, cotton, and moist bodies generally, and important insulators are resins, shellac, amber, caoutchouc, dry air, silk, glass, wax, and sulphur. A *dielectric* is a nonconducting or insulating material between two charged bodies.

Nature of Electricity.—The modern view of the nature of electricity is a return to an old theory qualified by recent scientific discoveries. According to this view, electrical phenomena are dependent upon the vibrations of the subtile, continuous, and incompressible ether which pervades all space. "This electrical ocean, in which we and everything else are immersed, has been likened by Sir William Thomson to a mass of jelly which allows all bodies to pass through it without friction, which is perfectly fluid for steady forces, but rigid for infinitesimal vibrations, and, as water is contained in jelly, so is electricity contained in the ether. Electricity thus becomes a mode or manifestation of the ether, as heat is a mode of motion of material particles. Conductors are bodies which allow electricity to flow through them. . . . The insulators or dielectrics are like elastic or impervious partitions, but yielding masses, subject to strains when electricity is moved. As a general definition it may be said that *all transparent substances* (not fluids) *are insulators*, and that all opaque bodies are conductors." (McClure.)

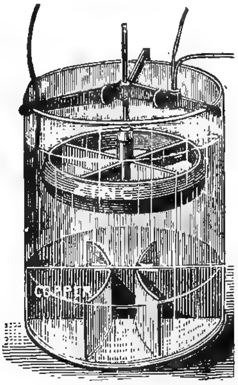
Galvanic Cells and Batteries.—Theoretically every substance, conductor or insulator, holds a certain electrical relation to every other, so that a list

FIG. 161.



Galvanic cell, showing position of negative plates and poles.

FIG. 162.

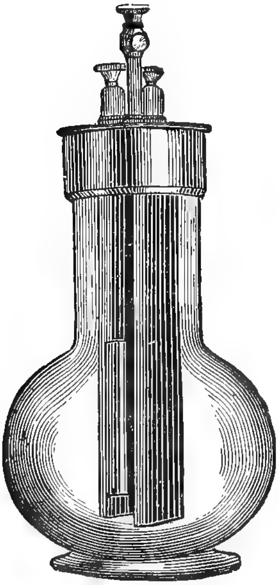


Gravity cell.

might be made in which each substance higher in the list than its fellows would be electropositive to all the rest. Zinc and copper,

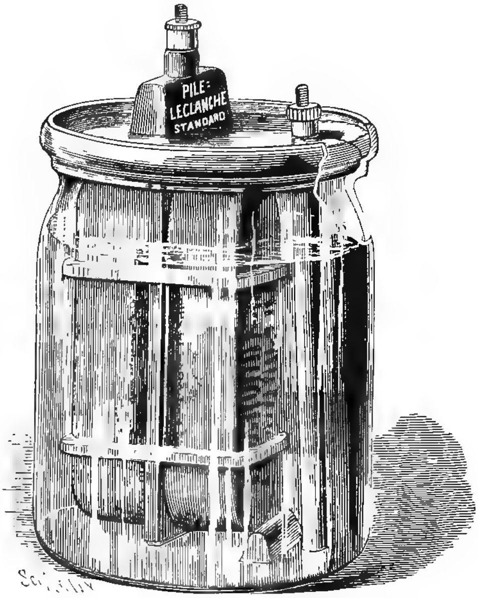
zinc and carbon, or zinc and platinum, are often used in the construction of batteries, the electropositive zinc being separated widely

FIG. 163.



Grenet cell.

FIG. 164.



Leclanché cell.

from the other elements named. The two metals are usually placed in a solution. The construction of a cell is seen in Fig. 161. A *dry cell*, so called, does not contain as much free liquid as a *wet cell*, moist material being used instead of the ordinary fluid. The absence of free liquid permits the cell to be closed. The separate cells of a medical battery should not be too powerful; the current supplied should be constant, should be comparatively smooth and regular, and should not flag during applications. Cells with one fluid are inconstant, but in medical practice some have been found to answer. For portable batteries hard rubber cells are preferable to those of glass or porcelain. Among desirable batteries are the Grenet (Fig. 163), Edison-Lalande, Law, Leclanché (Fig. 164), Gravity, and Chloride of Silver. The constituents of some of the best known batteries are shown in the following table. A great improvement in the Chloride of Silver battery, as formerly made, is in the use of what is known as the dry interchangeable cell (Fig. 165), the construction of which allows one or any number of exhausted cells to be disconnected and repaired, or new cells attached in their places.

FIG. 165.

Silver chloride dry interchangeable cell.

Constituents of Well Known Batteries.

NAME OF CELL.	POSITIVE ELEMENT.	NEGATIVE ELEMENT.	EXCITING FLUID.	DEPOLARIZER.
Daniell.	Zinc.	Copper.	Zinc sulphate.	Cupric sulphate.
Edison-Lalande.	Zinc.	Cupric oxide and carbon.	Potassium hydroxide.	Cupric oxide.
Gaiffe.	Zinc.	Silver.	Zinc chloride.	Silver chloride.
Gravity.	Zinc.	Copper.	Zinc sulphate.	Cupric sulphate.
Grenet.	Zinc.	Carbon.	Sulphuric acid and potassium dichromate.	Chromic acid.
Lalande-Chaperone.	Zinc.	Copper or iron.	Caustic potash.	Cupric oxide.
Law.	Zinc.	Carbon.	Ammonium chloride.	None.
Leclanché.	Zinc.	Graphite.	Ammonium chloride.	Manganese dioxide.
Walker.	Zinc.	Platinized graphite.	Sulphuric acid (dilute).	None.

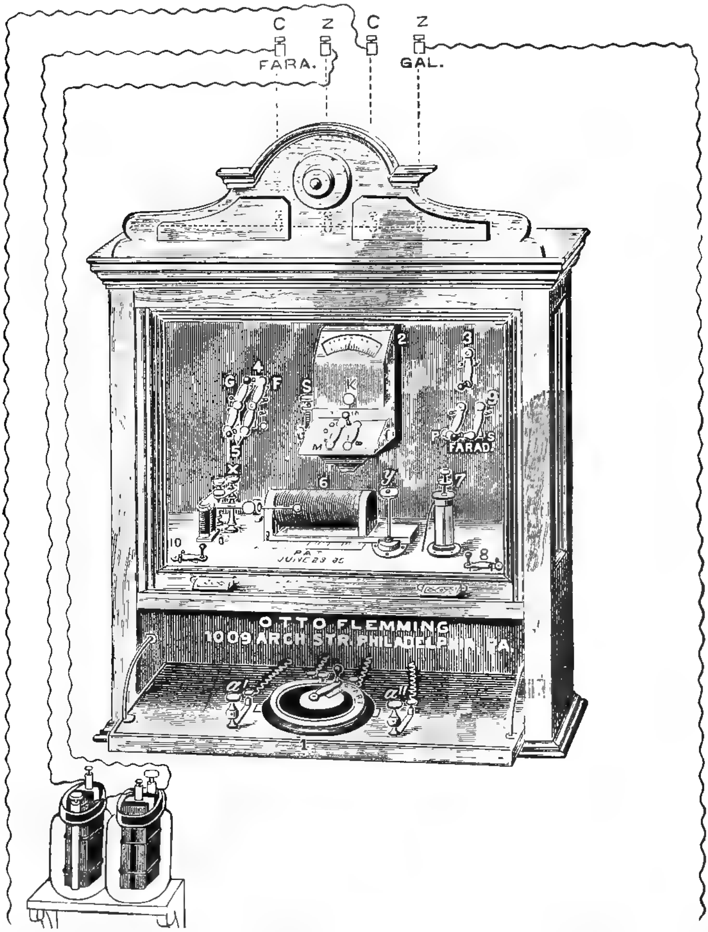
FIG. 166.

Galvanic battery, with silver chloride dry interchangeable cells.

Galvanic Apparatus for Medical Purposes.—The well known manufacturers of electromedical instruments all do work of such

high character that it is difficult to choose among them. Physicians will be well served by such firms as Flemming, Waite & Bartlett, the Chloride of Silver Dry Cell Battery Company, the Edison Manufacturing Company, the MacIntosh Company, the Galvano-Faradic

FIG. 167.



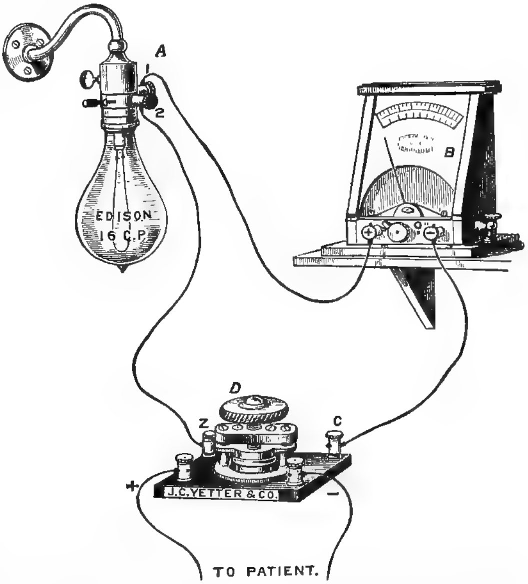
Electrotherapeutic wall cabinet: 1, Massey current controller; 2, Flemming milliamperemetre; 3, single switch for including or omitting the milliamperemetre in the galvanic circuit; 4, de Watteville current combiner; 5, the commutator or pole changer; 6, Du Bois-Reymond induction coil; 7, water rheostat; 8, single switch for omitting or including the water rheostat in the faradic current; 9, faradic double switch; 10, single switch for starting or stopping the faradic apparatus; x, contact screw; y, controlling screw; G, button for galvanic current; F, for faradic current; P, for primary faradic current; S, for secondary faradic current. For letters on milliamperemetre, see Fig. 172.

Company, and others. Illustrations are given of some of the best forms of portable and of permanent galvanic and faradic apparatus.

Apparatus for the Adaptation of Street Currents to Medical Use.—Since the general introduction of electric lighting, efforts

have been made to use street currents for medical work, and specially designed rheostats or adapters have been invented. The Vetter current adapter, which can be attached to a light fixture as readily as changing a lamp, is shown in Fig. 168, connected with a current controller and milliamperemetre. If connected with a sixteen candle power lamp, the rheostat will control the incandescent current from 0 to 500 milliamperes. To run a faradic coil, the current adapter is adjusted to a sixteen candle power lamp, and posts 1 and 2 are joined to an ordinary faradic apparatus, or an ordinary faradic battery can be united to the adapter. When the current is in use the lamp acts as resistance to the flow, allowing only a limited quantity of the current to pass in that circuit. While this and other forms of adapting apparatus have been recently much improved, many electricians believe that the street current cannot be safely tapped for medical purposes; certainly no one should attempt to make use of it without a knowledge of the principles involved, and of the possibility of accidents through carelessness or some unexpected defect.

FIG. 168.



The Vetter incandescent current tap: A, the current adapter, connected by a cord from post 1 with the milliamperemetre, B, and by a cord from post 2 with the controller D.

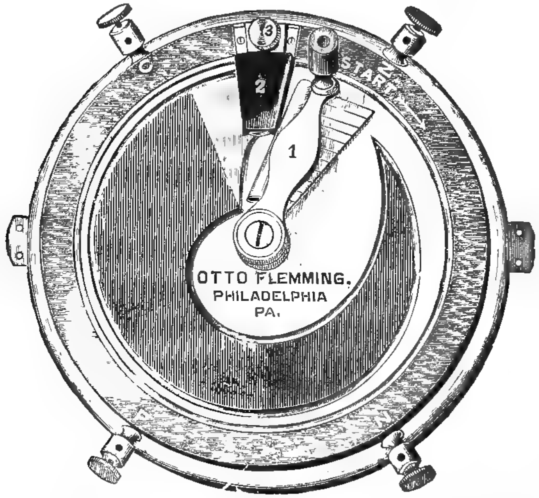
Apparatus for the Continuous Application of Weak Currents.—Many forms of electric disks, belts, chains, and girdles have been used, but most of these appliances are useless, as frequently no current is derived from them, or when one can be obtained it is too feeble to be effective. To make long continuous applications of electricity of small dosage, Dana has had constructed a small box apparatus, made by Stammer, of New York, which can be applied for six hours or more daily, and from which a continuous current of one-half to one milliampere may be obtained.

Rheotome.—A rheotome is an apparatus for interrupting the current; it may be a contrivance run by clockwork or electricity, or a special form of hand electrode. Duchenne invented a *pedal* or *foot rheotome* for slow intermissions, and Gowers has improved

it by having it so constructed that a slight twist of the foot will fix it immovably when required, even when the foot is removed.

Rheostats and Current Controllers.—The rheostat is an instrument by which the strength of a galvanic current can be regulated with exactness by increasing or decreasing the resistance without opening the circuit. Many forms of *wire rheostat* have been used in the arts and in medicine. It is only necessary to remember that the longer the wire through which the electric force is acting the weaker will be the resulting current. Another contrivance for diminishing coarse currents is the *water* rheostat, a glass tube with metallic bottom and top, in which works a movable rod. By the Massey *current controller* (Fig. 169) the current can be varied at will from a fraction of a milliampere to the full strength of the battery without shock, by rapidly increasing or decreasing the resistance of the circuit. It consists of a porcelain plate provided with a sickle shaped conducting area of graphite (soft pencil), broadening and thickening up to the terminal, where the graphite unites with a metallic surface which is in direct contact with the battery without any material resistance. The construction of the Vetter carbon current controller, shown in Fig. 170, is based upon the effect of the variation in resistance taking place in a quantity of carbon subjected to change of pressure. Finely divided carbon is placed in a small rubber cylinder between two metal plates which form the opposing ends of a circuit. The lower one is fixed to the base of the instrument, and the upper can be depressed by a fine screw, so as to compress the carbon. The government of the circuit is absolutely reliable, as the carbon resistance is susceptible to the slightest variation of pressure of the screw, so that any degree from an almost imperceptible current to the full power of the battery can be obtained. Equal work is imposed on the cells of the battery, and a current of uniform and equal strength can be maintained. Other advantages are the saving of space and the avoidance of annoyance in the

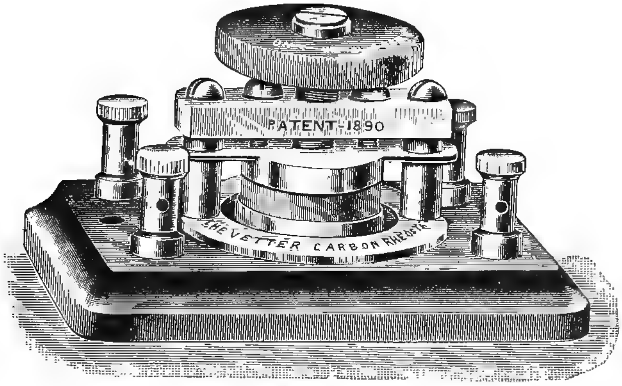
FIG. 169.



The Massey current controller: C, binding post to connect with carbon terminal; Z, binding post to connect with zinc terminal; N, anode or negative pole; P, cathode or positive pole; 1, turning crank; 2, hard rubber bridge.

absence of a complicated system of wires, and the absence of glass and liquid. Another excellent instrument is Willms's unalterable

Fig. 170.

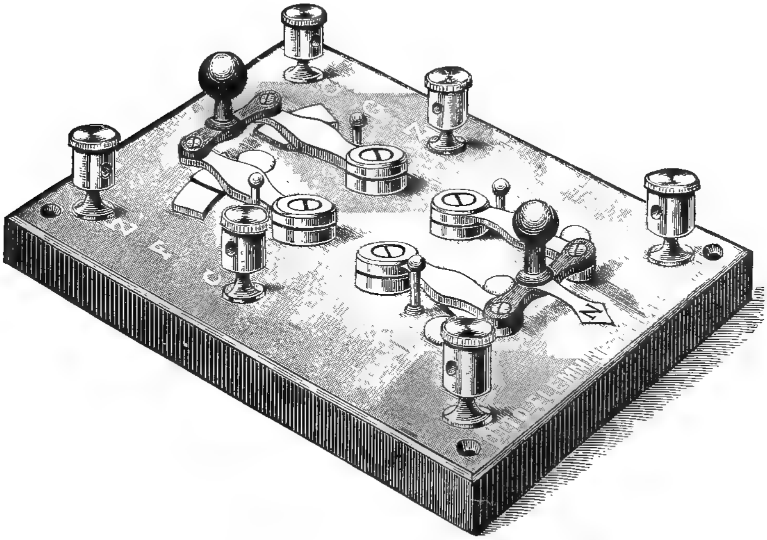


The Vetter carbon current controller.

current controller, in which the resisting material consists of common glue, graphite, and metal filings, the disk having a series of metal contact points with which the turning crank makes connection.

Commutators.—By a *commutator* or *pole changer* not only can the galvanic current be made and broken, but its polarity can be changed

Fig. 171.

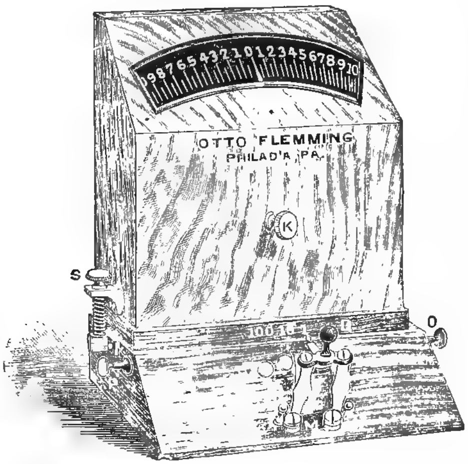

The de Watteville current combiner and commutator.

from moment to moment. A commutator may be a device on the switch board of the battery, or a foot or a hand instrument. In Fig. 171 is shown an apparatus devised by de Watteville, as both *current*

combiner and *commutator.* Both galvanic and faradic currents may be elicited simultaneously by placing the double switch in a parallel position. The faradic apparatus is started with all the switches set for actual work. The two currents are thus regulated each by their own controllers. For using the galvanic current alone, the upper double switch is moved so as to rest one part of the double switch on the flat plate G, and the other on the button in the middle. For the faradic currents alone, the double switch is moved so as to rest on F. The lower double switch is the commutator, with its index, N, always pointing to the cathode or negative pole. Gowers has invented a pedal commutator. Changing the polarity is more under the immediate control of the physician when it can be done by means of an apparatus held in the hand, and excellent pole changers of this kind have been invented by Birdsall and by Faught.

Electric Dosage.—Rules for electric dosage, applicable to all cases, cannot be formulated. An accurate galvanometer or a milliamperemetre (Fig. 172), divided according to an approved system, and electrodes of certain diameters, should be used. The so called Erb's normal electrodes, marked in square centimetres (Fig. 173) so that the current density may be known, have some advantages. With a uniform current strength the density is inversely proportionate to the diameter of the electrodes. The proportion which the current bears to its place of entrance and exit should also be considered. If a current strength equalled 6 milliamperes, and the electrodes 6×12 centimetres, or 72 square centimetres, the density would be equal to $\frac{6}{72}$ or $\frac{1}{12}$, which means that upon 12 square centimetres of electrode 1 milliampere of current is spread.* To express the cur-

FIG. 172.

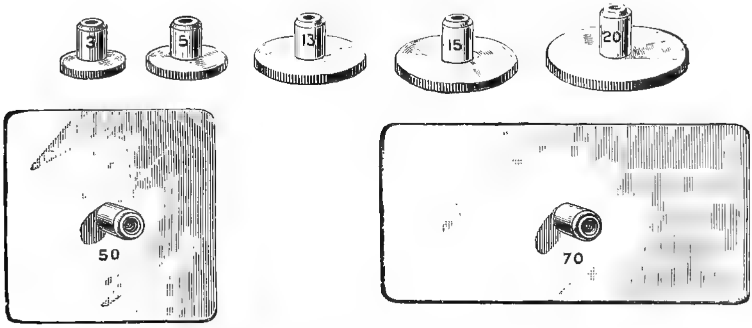


The Flemming milliamperemetre: O and P, binding screws to make connections for circuit. When the metre is being transported, the needle should be arrested by turning the knob K; if the needle, when released, does not settle at the 0 of the scale upon coming to rest, raise or lower the case by means of the adjusting screw, S, until the index is exactly at zero. The scale is divided into ten units from zero in either direction. When the double switch points to the figure 1 in front of it, the figures of the scale indicate a milliampere each. If the switch be moved to button 10, the scale reading is to be multiplied ten times, and if it points to 100, it is to be multiplied one hundred times.

* These suggestions are condensed from the recommendation made in 1886 by a committee of the American Neurological Association, consisting of Drs. G. W. Jacoby, W. F. Birdsall, and R. W. Amidon.

rent a fraction can be used, of which the numerator represents the number of milliamperes, and the denominator the number of square

Fig. 173.

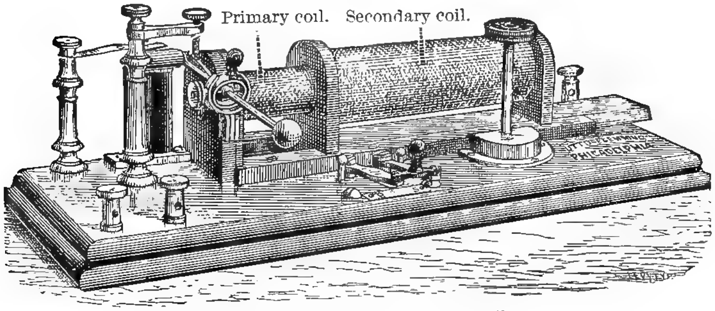


Erb's electrodes of regulated dimensions.

centimetres contained in the electrodes. Conclusions drawn from the number of cells employed cannot be depended upon, as the strength of a given number of cells is a varying quantity.

Faradic Apparatus.—Some faradic machines are made with several coils, but most of them have two, a primary and a secondary, as shown in the Du Bois-Reymond coil (Fig. 174). A faradic battery should be provided with a commutator or polarity changer,

Fig. 174.

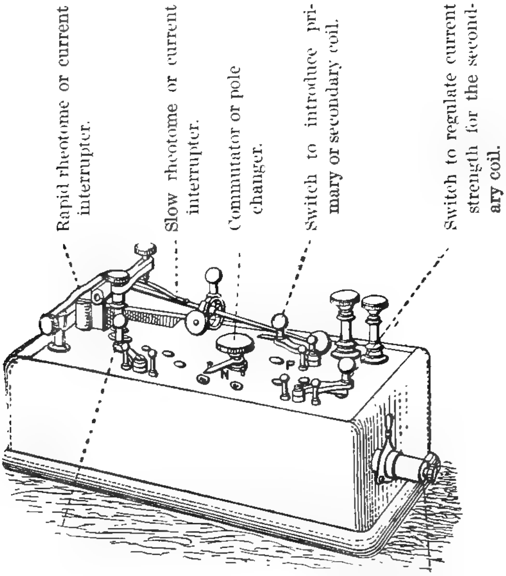


The Du Bois-Reymond induction coil.

with a slow and rapid rheotome or current interrupter, with a scale by which the currents may be finely graduated (Fig. 175), and with an enduring form of galvanic cell. In most faradic machines the first or inner coil consists of comparatively coarse insulated copper wire, one layer wound over another, and a second or outer coil of ten to fifteen times as much insulated wire of the size of very fine packthread. A standard faradic machine was adopted at the Electrical Congress in 1881. The galvanic generator is a single Daniell cell. The resistance in the primary coil is about 1.5 Siemens units;

in the secondary coil the resistance is about 300 Siemens units. (Robinson.) To gauge the faradic current, a scale is attached to

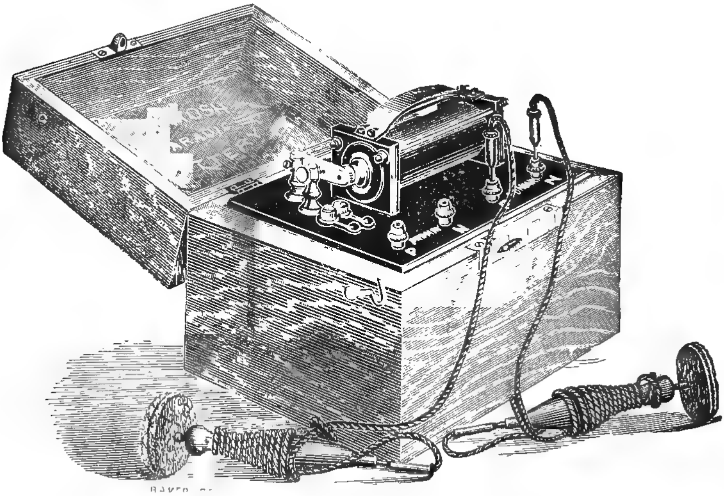
FIG. 175.



Faradic apparatus for office table.

the coil to indicate the strength of the current when the secondary

FIG. 176.



McIntosh's faradic battery.

coil is made to slide over the primary coil, the distance through which it is pushed being marked in millimetres

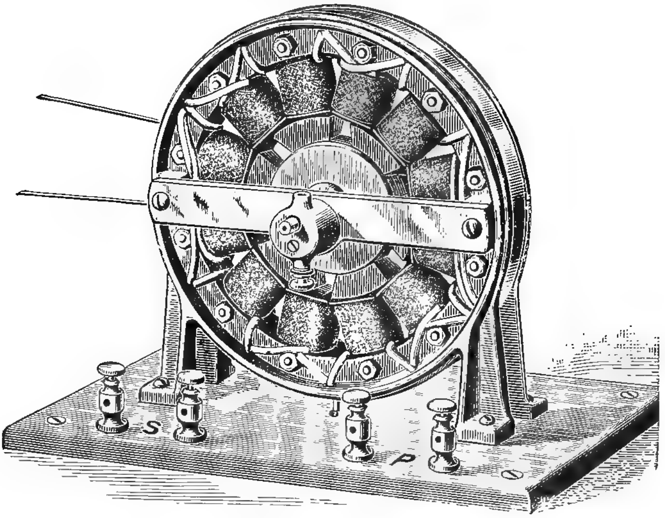
The various parts of the standard machine are as follows:

	Primary coil.	Secondary coil.
Length of spool (excluding wooden frame) . .	88.00 mm.	65.00 mm.
Diameter of spool	36.00 "	68.00 "
Diameter of wire	1.00 "	0.25 "
Number of turns of wire	300.00 "	5000.00 "
Layers of wire	4.00 "	28.00 "

The best method of marking a faradic current is to have the scale read zero when the secondary coil is removed from the primary.

Sinusoidal Current and Therapeutic Alternator.—The sinusoidal current, so named by d'Arsonval, on account of the sinusoidal form of the curve produced by the current when graphically represented, is obtained from a magnetoelectrical apparatus, like the Kennelly therapeutic alternator* (Fig. 177), which can be driven by a motor from a battery of eight Edison-Lalande cells or from a small motor or Edison incandescent circuit. It has twelve poles, on each of which is a spool with two windings of wires, the inner having eight layers of fine and the outer two of coarse wire. The former are connected in one series constituting the secondary or delivery coil, while the latter, connected in another series, form the primary or field winding of the machine. To transform the battery or continuous primary current into an alternating current wave in the secondary circuit, it is necessary to drive the armature, which is a combination of iron disks. By duly proportioning the grooves and projections in the armature surface the waves are made practically

FIG. 177.

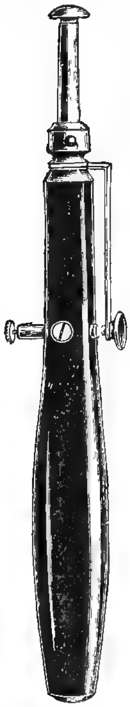


The Kennelly therapeutic alternator.

* Manufactured by the Edison Manufacturing Company.

sinusoidal. A rheostat of wire for battery circuits, or of lamps when operated from incandescent mains, is included in the primary circuit of the alternator, by which the strength of the secondary currents can be controlled independently of frequency. Special therapeutic effects can be obtained from using such a rapidly alternating or continuous sinusoidal current.

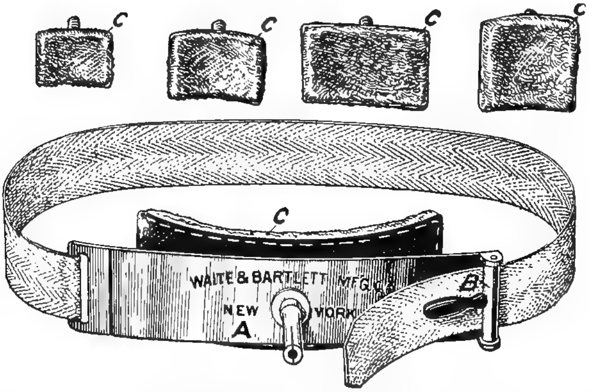
FIG. 178.



Interrupting hand electrode.

Electrodes.—It will not be necessary to describe in detail the different forms of electrodes, two of which are shown in Figs. 178 and 179. A neat method of covering an electrode is to employ absorbent cotton instead of chamois skin or sponge. (Massey.) After a time an electrode tends to become encrusted with salt, dirt, or chemical deposits, but one that is not clean should not be used. The deposits may cause local electrical reactions, and render the applications unnecessarily painful, and a clean polished electrode is therefore less painful than any other. When a current passes between a metal and a liquid electrolyte, a great apparent resistance occurs, the current becoming weaker, this diminution in the strength of the main current being due to a local action, called polarization, which gives rise to secondary currents, which act in an opposite direction to the chief current. Not only may the plates or elements of the battery become polarized, but polarization may occur at the surface of the electrodes, interfering with their efficacy. Unpolarizable electrodes have been invented by Du Bois-Reymond, but have not come into general use. Electrodes which can be adjusted to the head, spine, or limbs are often useful in neurological work. A good form of head electrode is shown in Fig. 179.

FIG. 179.



Putnam's head electrodes (*C, C, C, C, C*), held in place firmly by a belt (*A*) and spring (*B*), that can be used on the head or any part of the body.

ELECTRODIAGNOSIS AND ELECTROPROGNOSIS.

Diagnostic Uses of Electricity.—As an aid to diagnosis electricity is chiefly used to assist in differentiating central from peripheral paralyses. In malingering it sometimes has a definite value, because of its power to distinguish an organic peripheral paralysis from a simulated loss of power; but it is not true that the presence of normal response both to galvanism and to faradism indicates that loss of power is not real, for, as will presently be seen, in central palsies the electrical reactions are often practically the same as in health. Both faradic and galvanic currents can be used to assist in determining whether a painful spine is due to organic disease of periosteum, bone, cartilage, or membranes of the spinal cord, or whether it is merely assumed or is part of a hysterical or neuromimetic syndrome. Electricity may aid in the diagnosis of the presence of death through failure of the patient to respond to both sensory and muscular tests.

Central and Peripheral Paralyses.—A peripheral paralysis may be caused by a lesion situated in the muscles or in the nerve endings in the muscles, in the nerve trunk entirely outside of the cranium or spinal canal, in the root ganglia, in the foramen through which the nerve passes, in the intracranial or intraspinal course of the nerve, or even in the nerve roots and tracts at their nuclei of origin; and when the lesion is in any of these places the electrical response is peripheral. Similarly a central paralysis may be due to a lesion in at least half a dozen places between the nerve nuclei and the brain surface, as in the upper pons, or in the crus, capsules, alba, or cortex. In the columns of the spinal cord, a lesion which produces paralysis, if situated above the centres in which originate particular nerves which supply the paralyzed muscles, gives a paralysis which is central for these nerves and muscles, although the same lesion might cause a peripheral palsy of the nerves originating in its immediate vicinity. In peripheral motor paralysis, electrocontractility both to faradism and to galvanism is usually lost wholly or in part, but in some cases, on account of the slightness of the lesion, the electrical changes cannot be recognized. In true central paralyses, whether spinal or cerebral, electrocontractility is not lost, although occasionally it is quantitatively depressed. If a lesion is at the same time both central and peripheral the responses to the electric current will be peripheral. Illustrations of central lesions are to be found in cerebral hemorrhage, embolism or thrombosis, tumors or abscesses, and some forms of sclerosis. Myelitis of various types will give central or peripheral reactions according to the diffusion of the lesion. Diseases which give diagnostic peripheral reactions are facial paralysis, traumatic paralysis, diphtheritic and lead paralysis, and other forms of palsy due to infectious or toxic agents.

Reaction of Degeneration.—The *reaction of degeneration* is a marked change in excitability or contractility observed when neuromuscular examinations are made with the faradic and galvanic currents. Coincidently with more or less wasting, faradic contractility may be entirely abolished, while to galvanism certain peculiar changes, which are spoken of technically as *modal* or *serial*, may be present. The modal change shows itself to the eye of the investigator as a slow, wormlike quivering of the degenerated muscle, even under the influence of a very weak current. Once seen and understood it cannot well be forgotten. Serial changes are qualitative also, and the term is used because when the anode or cathode is successively applied on opening or closing the circuit we get a series of differences in the degree of response and, to some extent, in its character. When nerve and muscle are normal this formula takes a certain order, the cathodal closure leading, the anodal closure coming second, and then the anodal and the cathodal opening. This order is changed in degeneration reaction. In forms of disease in which nerve and muscle slowly degenerate, these tissues gradually grow more and more abnormal in their response to both galvanism and faradism. This gives what is called *partial degeneration reaction;* it is easily determined when it is remembered that both nerve and muscle continue to respond to galvanism and faradism as long as any nerve fibre or muscular tissue remains. In the *complete* form of the reaction of degeneration the muscles, while showing lost irritability to faradism, may show an increased irritability to galvanism. In partial degeneration reaction easily recognized modal changes are present, and polar changes can be determined, but may require careful and skilful investigation. The degeneration reactions are dependent upon pathological changes which occur in nerve and muscle. As regeneration progresses the electrical reactions become more and more those of health. In old cases of paralysis, nerve and muscle sometimes entirely disappear, and no reaction can be obtained with any form of electrical current. Technically, this is not a reaction of degeneration, but simply an absent reaction. A reaction of degeneration which is at one time partial may in the downward progress of the disease become complete, and the same muscle, when large and composed of separate bundles, may even show differences of reaction in different parts.

Symbols and Formulas.—The following symbols are sometimes employed in studying these reactions:

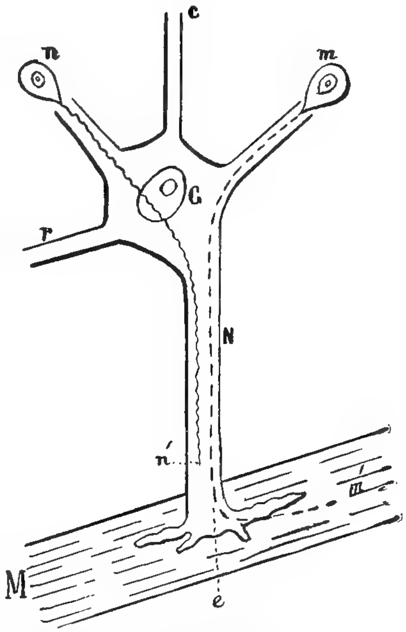
An, anode.
Ca, cathode.
De R or RD, reaction of degeneration.
C, contraction.
Cl, closure.
O, opening.

CaClC, cathodic closing contraction.
CaOC, cathodic opening contraction.
CaClTe, cathodic closing tetanus.
AnClC, anodic closing contraction.
AnOC, anodic opening contraction.
AnClTe, anodic closing tetanus.

Using symbols, the normal reaction is expressed by CaClC > AnClC > AnOC > CaOC. Anodal closure contraction may equal cathodal contraction, as AnClC = CaClC. When reaction of degeneration is present, the anodal opening contraction may lead the series, giving AnOC > AnClC > CaClC > CaOC, or it might be properly represented as follows: AnOC > CaClC = AnClC > CaOC. Some of these varieties in reaction of degeneration occur so regularly as to be regarded as typical forms; for instance, where AnClC > CaClC, or AnClC = CaClC.

Independence of Trophic and Paralytic Phenomena.—According to de Watteville, trophic changes in muscle may be independent of paralytic phenomena, and may affect both muscle and nerve or muscle alone. Fig. 180 shows the relations between the motor and the assumed trophic centres, and between the nervous and muscular fibres. The trophic as well as the voluntary and reflex influences are supposed to converge in the multipolar ganglion cell, G. If the lesion be at *c*, or in the cerebral centres above, the characteristic symptom is paralysis (loss of motility), but there is no atrophy nor loss of electroexcitability. A lesion involving both *c* and *m* causes paralysis with atrophy of muscle, but not of nerve, and gives qualitative and quantitative changes in galvanomuscular irritability, the nerve responding to both galvanism and faradism, as in amyotrophic lateral sclerosis. If the lesion is confined to the trophic centre (*m*), the muscle atrophies, but is not paralyzed, and presents qualitative alterations in reaction. Electronervous excitability is present. The phenomena are those of partial RD, and occur in progressive muscular atrophy, bulbar paralysis, mild acute poliomyelitis, etc. "When G is destroyed, there is loss of motility, loss of reflex action, atrophy both of nerve and muscle. The electrical phenomena are those of the reaction of degen-

Fig. 180.



Erb's diagram, showing the relations between the motor and assumed trophic centres and the nervous and muscular fibres: M, muscular fibre; N, nerve fibre with its ending, *e*; G, multipolar ganglion cell, from the anterior horn of gray matter or bulbar nuclei; *c*, path of impulse from the brain, anterolateral columns; *r*, path of reflex excitation from the sensory sphere; *m*, trophic centre for the muscle; *n*, trophic centre for the nerve; *m–m′*, path of trophic influence to the muscle; *n–n′*, path of trophic influence to the nerve. (De Watteville.)

eration fully developed. The same phenomena must obviously be present when N is destroyed at one point completely (*i.e.*, including m' and n'). G is destroyed in anterior poliomyelitis (infantile paralysis); N, in severe peripheral paralyses."

Table showing the Connection between Certain Pathological States and their Electro-diagnostic Phenomena. (Modified from de Watteville.)

SEAT OF LESION AND DISEASE.	PROMINENT SYMPTOMS.	ELECTRICAL REACTION.
Cortex and pyramidal tracts (c). Cerebral hemorrhage, embolism, thrombosis, tumors, abscess, and lateral sclerosis.	Paralysis without muscular degeneration; contractures.	Normal.
Pyramidal tracts; ganglion cells of ventral horns of the cord (c and m). Amyotrophic lateral sclerosis.	Paralysis and muscular degeneration; contractures.	Nerve normal; partial RD in muscle.
Trophic centres for muscle to cells of ventral horns of cord, or to bulbar motor nuclei (m extending to G). Progressive muscular atrophy of central origin; bulbar paralysis; mild acute poliomyelitis.	At first no paralysis; later paralysis and muscular followed by nerve degeneration.	Nerve normal; partial RD in muscle.
Multipolar ganglion cells (G). Anterior poliomyelitis.	Paralysis and atrophy of nerves and muscle, with abolition of reflexes.	RD for nerve and muscle.
Nerve and muscle (N, n' and m'). Rheumatic, traumatic, or toxic disease of nerve and muscle.	Paralysis and muscular degeneration.	Partial or complete RD for nerve and muscle.
Muscle (M). Muscular wasting in phthisis, diseases of joints, idiopathic myositis; primary myopathies.	Paresis and simple atrophy.	Gradual diminution of electrical response.

Peculiarities in the Electrical Reactions.—In many forms of neuritis, and especially in that due to lead poisoning, nonparalyzed nerves and muscles may show changes in electrical irritability indicating the presence of unsuspected degenerative processes. (Gumpertz.) Putnam found that even powerful and painful currents failed to cause contraction of the extensors of the finger when the anode of the faradic battery was applied to the musculospiral nerve, although the response to the faradic current and that to the direct excitation of the extensor muscles were apparently normal, but almost the same results were obtained with normal subjects. The reactions in multiple neuritis are worthy of special mention. They vary in different paralyzed muscles, as would be expected, because of the local variations in the intensity of a multiple disease. Sometimes polar changes are not present, although the response to both currents may be diminished; sometimes with total loss of faradocontractility no galvanic polar changes can be determined; and sometimes electrical changes are present in unparalyzed muscles. Polar irritability will also vary much at different stages of such a disease as poliomyelitis.

Electrodiagnostic Technique.—Some points to be observed are (1) to make the patient as comfortable as possible; (2) to see that the muscles are relaxed and at rest; (3) thoroughly and equally to moisten the electrodes; (4) to pass the current through equal lengths of the two parts compared; if one side is compared with the other, by making one large electrode stationary in a central region, as the sternum or sacrum, and applying the other smaller electrode to the nerve or muscle; (5) to begin the tests on the healthy part, determining first the least strength of current that will cause contractions, and then carefully to make the test in exactly the same way on the diseased part. Usually the nerve and muscle are first tested with the faradic current.

FIG. 181.

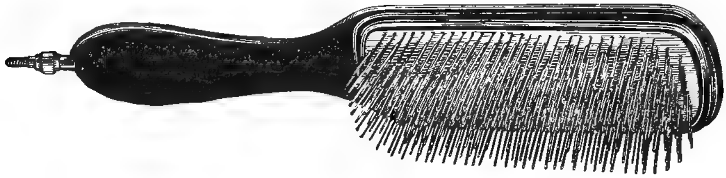
Leszynsky's diagnostic electrode: the handle is so constructed that the circuit can be opened and closed without removing it from the skin; the connections are completely insulated, its shape adapts it to the operator's fingers and hand, and the curve on the shaft attached to the motor point facilitates its adjustment and manipulation.

The Myotonic Reaction.—To obtain the myotonic reaction—a peculiar form of electrical response observed in Thomsen's disease—a large electrode is placed upon the sternum or upon the back of the neck, and another of medium size in the palm. Allowing a medium current to flow, a tonic spastic condition of the muscles of the arm is developed. In a little while, particularly after changing the poles with the commutator, curious wave-like contractions take place, these moving downward when the anode is in the hand, upward when the cathode is in the same position. (Jacoby.) Erb has compared the single waves to those produced by a stone falling in water. The amount of current requisite for the production of the phenomena varies from six to twenty milliamperes. Erb asserts that this reaction is always characteristic, but that it may be found in normal and nearly normal muscles.

Electrosensibility.—The electrosensibility of the skin, sometimes important in the diagnosis of hysteria or malingering, is determined by faradization with a metallic brush (Fig. 182) or with other metallic electrodes. Two parts of the body can be compared, or the application can be made to the same area in two individuals. The strength of the current which excites the first trace of sensation, and also that which causes distinct impressions of pain, should be noted. Leyden's method of using compasses connected with a faradic apparatus may be employed. In any method provision can be made for reading off the distance separating the

ends of the induction apparatus. One electrode is placed upon the portion to be examined, the other, or indifferent electrode, may be applied anywhere. Averages are obtained for different parts of the body, and these enable deviations from the normal to be detected.

Fig. 182.



Brush electrode.

Bernhardt has divided the surface of the body with reference to cutaneous electrosensibility into nine zones. These and the average minimum coil distance of sensation and of pain for these various regions are given in the following table (combined from two tables by Robinson):

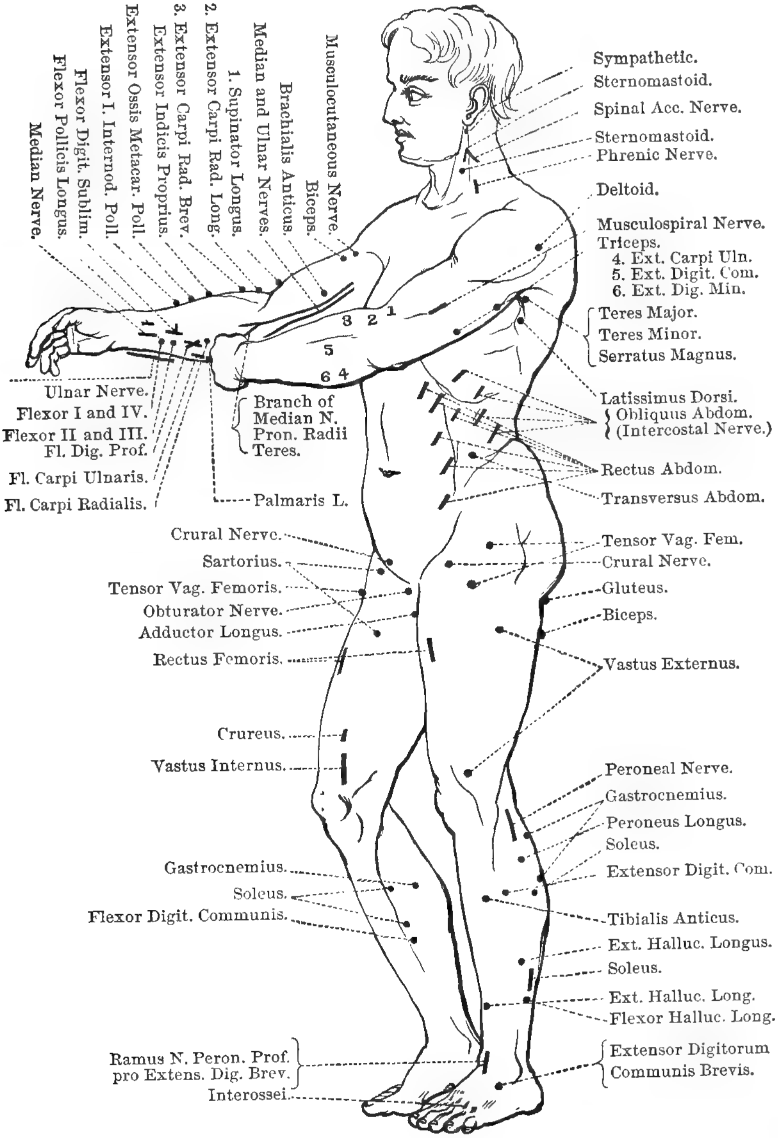
Zones.	General Electro-sensibility.	Painful Electro-sensibility.
1. Tongue (tip of tongue, palate, tip of nose)	166.0 mm.	136.7 mm.
2. Face (eyelids, gums, red lips, cheeks)	150.5 "	130.5 "
3. Forehead	144.5 "	128.0 "
4. Shoulder	137.0 "	112.5 "
5. Trunk (sternum, nape and front of neck, occiput, spine, arm, forearm, buttock) .	128.0 "	110.8 "
6. Thigh (sacrum, thigh, dorsum of foot)	122.1 "	99.1 "
7. Hand (back of hand, leg, ball of fingers) .	116.0 "	92.8 "
8. Patella	111.0 "	88.0 "
9. Digital (palm, tip of toes, sole of foot) . .	114.5 "	67.8 "

Electroprognosis.—The value of electricity in prognosis—mainly in the prognosis of paralyses—has been largely foreshadowed in the remarks on electrodiagnosis. Having determined that a paralysis is central, the gravity of the prognosis is made evident; and, on the other hand, if the diagnosis has been decided in favor of a peripheral lesion, although the electrical changes are more serious, the prognosis is comparatively good. An understanding of the subject of partial degeneration reactions may help to settle the cornual nature of a progressive atrophic disease, and, in so doing, decide for it a serious prognosis and a chronic course. As time advances in the treatment of certain peripheral paralyses, as Bell's palsy, the prognosis becomes more and more certain as the electrical reactions change for better or worse. Step by step in some of these cases the stages of degeneration can be marked by the sinister changes in electrical response until finally no reaction whatever can be obtained. Faradic contractility may be entirely lost for a time in cases with comparatively favorable prognosis.

ELECTROTHERAPEUTICS.

Special Methods of Electrical Application.—A current is *direct* when the anode is applied at or near the neuraxis and the cathode

Fig. 183.

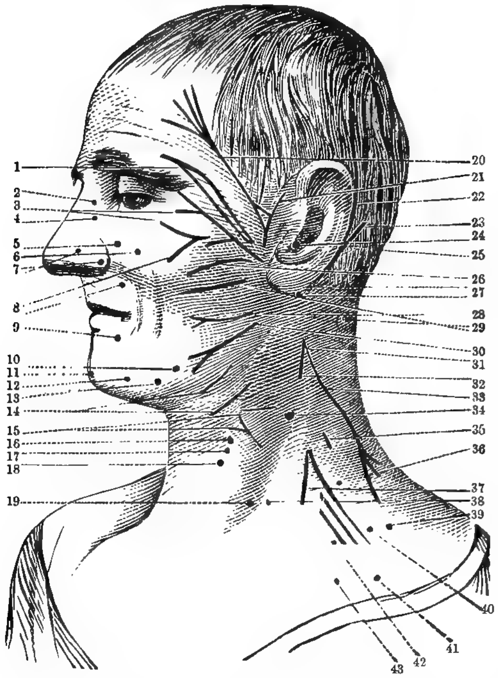


Motor points of trunk and limbs. (Lincoln.)

towards the periphery, while the reverse positions of the electrodes give an *indirect* current. In a *unipolar application* the anode or the cathode is applied to a selected spot, and the other electrode is placed

upon a part so distant that the effects of the two poles cannot commingle. In a cutaneous application both the skin and at least one electrode should be dry. Applications can also be made to the skin by the dry or slightly moistened hand. For deeper seated applications to motor nerves and muscles the electrodes should be covered with some substance like absorbent cotton, sponge, or chamois skin, which should be thoroughly wetted with warm water or warm salt water. Firm pressure should be made over the motor point or muscle to be reached. For paralysis either *direct* or *indirect muscular electrization* may be used; in the former the electrodes are applied as directly as possible to the muscles; in the latter the muscle is caused to contract through its nerve supply, by applying the electrodes to *motor nerve points.* Motor points, nerves, and important muscles are shown in Figs. 183, 184. Joint applications can be made by applying electrodes to opposite sides of the joint, or by placing one electrode, with the foot or hand, in a basin of warm salt water, while the other is applied to some portion of the limb. For the *eye*, a moistened rheophore is applied to the closed eye, and a second to any convenient spot; for the *ear*, a

Fig. 184.



Points for muscles and motor nerves of the face: 1, m. corrugator supercilii; 2, m. compressor nasi et pyramidalis nasi; 3, m. orbicularis palpebrarum; 4, m. levator labii superioris alæque nasi; 5, m. levator labii superioris (proprius); 6, m. zygomaticus minor; 7, m. dilatori naris anteriori et posteriori; 8, m. zygomaticus major; 9, m. orbicularis oris; 10, ramus communicans pro m. depressor anguli oris (triangularis menti) et levator labii inferioris (levator menti); 11, m. levator labii inferioris (levator menti); 12, m. depressor labii inferioris (quadratus menti); 13, m. depressor anguli oris (triangularis menti); 14, ramus subcutaneus colli n. facialis; 15, ramus cervicalis pro m. platysma myoides; 16, m. sternohyoideus; 17, m. omohyoideus; 18, m. sternothyroideus; 19, m. sternohyoideus; 20, m. frontalis; 21, m. attrahens aurem et attollens aurem; 22, m. retrahens aurem et attollens aurem; 23, m. occipitalis; 24, n. facialis; 25, ramus auricularis posterioris pro n. facialis; 26, m. stylohyoideus; 27, m. digastricus; 28, rami buccales n. facialis; 29, m. splenius capitis; 30, ramus subcutaneus maxillæ inferioris; 31, ramus externus n. accessorii Willisii; 32, m. sternocleidomastoideus; 33, n. accessorius; 34, m. sternocleidomastoideus; 35, m. levator anguli scapulæ; 36, n. thoracicus posticus (m. rhomboidei); 37, n. phrenicus; 38, m. omohyoideus; 39, n. thoracicus lateralis (m. serratus magnus); 40, n. axillaris; 41, ramus plexi brachialis (n. musculocutaneus pars n. mediani); 42, 43, n. thoracici anteriores (m. pectorales).

medium sized electrode is placed so as to cover the tragus, and the indifferent pole to the hand or the sternum; for the *nose*, a moist rheophore is applied to the back of the neck, and a metal sound, insulated except at its extremity, to the nasal mucous membrane; for the *tongue*, one electrode is applied to the neck, while the other is moved over the base and border of the tongue.

Care and Method.—Electricity sometimes falls into unmerited disrepute because of the extreme carelessness and want of interest with which it is used. It is better that applications should be frequent rather than severe and long continued. As to the duration of a single application, it is difficult to give fixed rules. Lincoln suggests as an average time three to five minutes to the spinal cord, one to three minutes to the organs of sight and hearing, two minutes to a single large muscle, five minutes to a group of muscles, and twenty minutes for general electrization. Incautious applications may produce vertigo or even syncope or faintness, results probably brought about through direct or reflex effect upon the pneumogastric nerve. Various annoying phenomena, as local or general rashes, blisters or burns of the skin or mucous membrane, and pain in the skin and muscles, may occur. If the muscles and coarse nerves of the trunk and limbs can be exhausted by electricity, much more certainly can the delicate retina be exhausted and its functions impaired or destroyed by this agent; and the nerves of hearing, smell, and taste can likewise be injured by overstimulation. Ritter, through experiments performed upon his auditory nerves for scientific purposes, destroyed his sense of hearing.

Electricity in Neurological Therapeutics.—Electricity is of value in the treatment of neurasthenia, hysteria, and paralytic and other diseases of the peripheral nerves, also for the relief of pain, particularly when of neural origin, for sleeplessness and headache, for a few affections of the spinal cord, brain, and nerves of sense, and for occupation neuroses.

General Faradization.—General faradization, which is most used in the treatment of neurasthenia and hysteria, is usually applied in either a reclining or a sitting position. (1) *In the reclining position,* beginning with the limbs, preferably the lower extremities, the electrodes are applied either directly over the muscles or to the motor points, care being taken to include as far as possible every muscle and group of muscles. The operator passes from the toes and feet to the legs and thighs, taking each set of muscles in turn. The upper limbs are next faradized, beginning with the fingers and hands and ascending to the shoulders, and the muscles of the back, chest, and abdomen are treated last, although it may sometimes be advisable to treat the muscles of the back, loins, and abdomen before the upper extremities. Violently contorting the muscles should be carefully avoided. From time to time the electrodes should be rewetted.

(2) *In the sitting position*, one method is to place in the patient's hand one of the well wetted electrodes; the other is held by the operator, who at first, with his free hand, makes applications to the head, face, and neck, after which, the patient still keeping one electrode in his hand, the operator passes the other down the back and arm. The patient then shifts the electrode to the opposite hand, and the operator passes down the arm of that side. Both electrodes can be taken into the hand of the operator and rapidly glided over the arms and trunk. The lower extremities are treated last. Another plan is to have one or both feet upon a large metallic electrode covered with a moistened cloth or chamois skin, while the operator passes the other carefully over all accessible portions of the body. Unnecessary exposure should be avoided. The operator should be satisfied with a moderate application. General faradization can be completed in from twenty to forty minutes. Treatment should not be too soon after meals, as it may interfere with digestion. The preferable time is midway between meals. The muscles should be relaxed during the application. When possible, both electrodes should be held in one hand, as shown in Fig. 185, as this leaves the other hand free to regulate the strength of the current or for other purposes. The electrodes are held pointing backward, the handle of one between the index and the middle finger, and that of the other between the ring and the little finger. The disks can be thus brought close together or separated several inches.

Fig. 185.

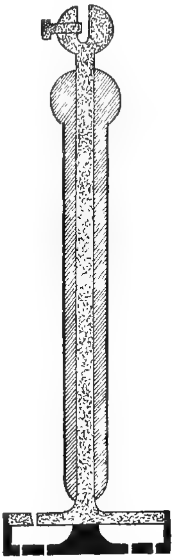
Method of holding electrodes in one hand.

Galvanofaradization.—The galvanic and faradic currents can be used simultaneously by a process called galvanofaradization, which may be either general or localized. It requires a double electrode with one part for each current. The copper plate may be connected at one part with the pole of the faradic and at another with that of the galvanic apparatus, and the circuit thus completed for both currents. In localized galvanofaradization it is necessary to have in use two double electrodes. By a proper construction and adjustment of the electrodes it is possible to localize the two currents very near to each other. (Beard and Rockwell.)

Cataphoresis.—Adamkiewicz first called attention to a method of treating pain in the superficial nerves by the application of chloroform to the affected area by means of an anodal diffusion electrode. The Ayres-Adamkiewicz electrode is shown in Fig. 186. The proximal end of the reservoir can be unscrewed and the whole interior exposed; and to allow of freer transudation of sufficient fluid

through the carbon plate several small apertures have been made. In this modified electrode a carbon column is made solid with a diaphragm abutting against a metallic plate. Peterson considers the Adamkiewicz electrode faulty on account of the metal cylinder with its carbon button. His electrode is shown in Fig. 187. When cataphoresis is employed the drug solution must be placed upon the anode, as the process is one of anodal diffusion, and the movement of the current carries substances in one direction only, from the anode to the cathode. Medicaments can be made to act by cataphoresis to reach morbid tissues, to produce absorption, as an alterative, to bring about local anesthesia, and to relieve pain in the superficial nerves. (Peterson.) Three methods of cataphoresis are (1) by the bath, (2) by solution of a drug on any ordinary sponge electrode, and (3) by the cataphoric electrode. Aconitia with cocaine, helleborin,—which should be used with caution,—ouabain, strophanthin, chloroform, and a weak solution of carbolic acid are efficient anesthetics. By *anemic cataphoresis* the drug is restricted to that part for which it is intended by cutting off the blood stream, either by an Esmarch bandage or a rubber ring, or by compression with a disk-shaped electrode. The medicine is incorporated in a small plaster composed of pulverized gas carbon and gelatin. (Morton.)

FIG. 186.



The Ayres-Adamkiewicz electrode.

FIG. 187.

Peterson's improved cataphoric electrode: *A*, disk made of metal that will not oxidize; the stem passing through the hard rubber cover, *C*, is held in place by the nut *D*, which holds the tip for connecting with the battery; *B*, soft rubber ring which is held in place by *A*, at the same time insulating the skin from *A*, allowing the current to pass from *A* to the skin through medicated paper contained in the cavity formed by *A* and *B*.

Treatment of Pain by Rapidly Interrupted Faradic Currents.—Dentists have long used the rapidly interrupted faradic current as an anesthetic. Some faradic instruments are made with separate induction coils, one with slow or moderate interruptions for the treatment of motor nerves and muscles, and the other arranged for exceedingly rapid interruptions in order to produce sensory effects. Hutchinson found that when the vibrations were kept so as to sound the note C the marked anesthetic effects of the current were produced and maintained. Tic douloureux has been entirely relieved and sleep induced by this method of treat-

ment, placing the negative pole under the nape of the neck and the positive—a flat sponge three inches square—upon the forehead. Kennelly and Peterson, at the Edison laboratory, investigated the effect upon themselves of a current alternating two hundred and eighty-eight times per second. They made the remarkable discovery that as the vibrations increased in frequency a benumbing effect was produced upon the sensory nerves, and with the highest number of vibrations a complete loss of sensation resulted in the parts in which the current was applied, so that pricking with needles and knives and the applications of cold and of hot water were not felt at all. When the current was turned off sensation was restored.

Voltaic Alternatives.—When an electrode on a nerve is changed from the anode to the cathode, and the reverse, a series of closure excitations are given, which fall for the cathode in the polar region and for the anode in the peripolar region respectively; and in either case the excited region has just before been under anodic influence. Physiology teaches us that the instant the polarizing current ceases to flow the anodic region passes into a state of increased excitability, and this augmentation is more marked the longer the anodic influence has lasted. *Voltaic alternatives* therefore act more powerfully than simple closures of the circuit, and their action is intensified by previous current duration. (De Watteville.) The use of voltaic alternatives is the most energetic method of electrical stimulation, and rapid reversals are more effectual than slow. This mode of treatment has been strongly recommended in forms of optic nerve atrophy by Fox, Faught, and Riggs. Voltaic alternations are frequently used by neurologists in the treatment of neuromuscular affections.

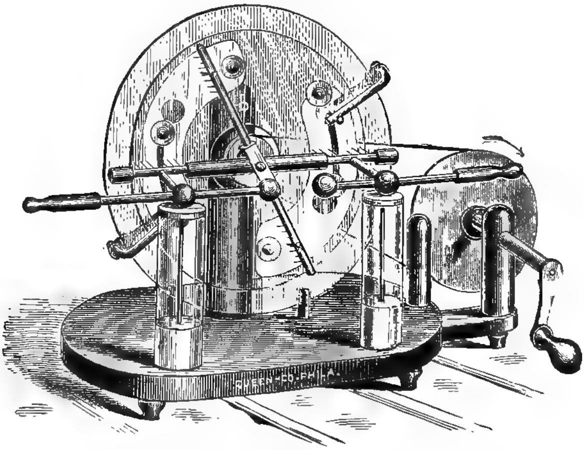
Miscellaneous Uses of Electricity.—Chronic poliomyelitis may make considerable improvement under the use of galvanism applied to paralyzed nerves and muscles, and a galvanic current of medium strength is a valuable remedy for chronic neuritis. Tremor, when functional or toxic, is sometimes greatly benefited by galvanism, applying the anode to the affected part, and the cathode to the spine or to some other indifferent spot. Chorea has been treated by all forms of electricity,—by sparks from frictional machines, by general and peripheral faradization, and by galvanization of the head and spine. Hysterical or nervous deafness, and some forms of tinnitus, may be successfully treated by faradism or galvanism. In treating tinnitus with hyperesthesia with galvanism, the anode is applied to the neighborhood of the ear, while tinnitus with anesthesia calls for the cathode to the ear or near it. A rheostat must be employed, or otherwise the unpleasant phenomena produced by the applications may compel the procedure to be stopped. Faradization is useful for those forms of tinnitus which depend upon paralysis

of the intrinsic muscles of the ear. In weakness of sight, when no lesion can be discovered—a condition which may be due to malnutrition, and a consequent oversusceptibility to fatigue—galvanic treatment with a weak current to the closed eyes is sometimes efficient. Hiccough, nervous asthma, and nervous vomiting are more or less amenable to electrical treatment by applying a weak current—the anode to the pneumogastric nerve, and the other electrode on some indifferent spot. Care should be taken not to place or remove the electrode abruptly, otherwise vertiginous attacks and even syncope may be produced. Writer's cramp is treated by passing a mild current in a descending direction through the nerves and muscles affected, at the same time exercising the muscles with light gymnastics. The anode may be placed in the axilla, and the cathode on the ulnar nerve at the elbow ; or the anode over the median nerve in the upper arm, and the negative over the flexor longus pollicis. (Poore.) The faradic current may, in some instances, be usefully conjoined with galvanism. Spermatorrhea, spermatophobia, and psychical or physical impotency may be benefited by the use of either the faradic or the galvanic current. Applications are made to the spinal column ; or one rheophore is placed upon the spine and the other applied to the perineum, or to the genitals, or to the urethra with an electrical bougie. Sleeplessness may be successfully treated by a weak galvanic current, placing flat electrodes on the temples for thirty seconds to one minute. Localized faradization is useful in hysterical aphonia, and in some cases of aphonia the result of organic lesion. Hemicrania during the attack is sometimes benefited by mild faradization of the painful region of the head, the application being made with the dry or slightly moistened hand. For cerebral neurasthenia weak galvanic currents to the head and general faradization, with rest and full feeding, have proved useful. Erb has strongly advocated the use of the galvanic current in the treatment of insanity. Cerebral galvanization, and the central galvanization of Beard and Rockwell, are used in mild mania, melancholia, and the early stages of general paralysis. In cerebral galvanization the electrodes are usually applied longitudinally, one to the occiput and the other to the forehead. The current should be weak, at first not appreciable, but slowly increased by the use of the rheostat or current controller ; the application should not be continued for more than three to five minutes. In central galvanization a weak current is used, and one electrode, usually the cathode, is applied to the epigastric region, while the other is placed first on the forehead, and then successively on the top of the head, along the sternocleidomastoid muscle in the neck, to the nape of the neck, and down the spine. The whole application should occupy from fifteen to twenty minutes. General faradization is a useful adjunct in the treatment of neurasthenic melancholia.

FRANKLINISM OR STATIC ELECTRICITY.

Franklinism and Franklinic Machines.—Static, frictional, or franklinic electricity, or franklinism, the first form of electricity used in medicine, was supplanted for a time after the discoveries of galvanism and faradism, was again brought into prominence in several eras, and was then relegated to obscurity; probably it has now received its proper position, although the tendency is still on the one hand to deny it any virtue, and on the other to claim for it too much therapeutic importance. Neither "static" nor "frictional" is a term altogether applicable to the description of this agent as now used. Underlying the construction of static machines are four principles,—that bodies may be electrified by friction; that they may be electrified by induction or proximity; that electrified bodies not only attract non-electrified bodies but communicate electricity to them by contact; and that bodies similarly electrified, either by each other or from the same source, show mutual repulsion. (McClure.) The first static or frictional machines were based upon the fact, known for centuries, that friction of substances like amber or glass by silk, and of resinous substances by flannel or catskin, would develop electricity of the so-called positive or negative character. A ball of sulphur was made to turn upon its axis and was grasped by the hands; later globes and tubes of glass and glass plates were used; still later, cylinders of glass

FIG. 188.

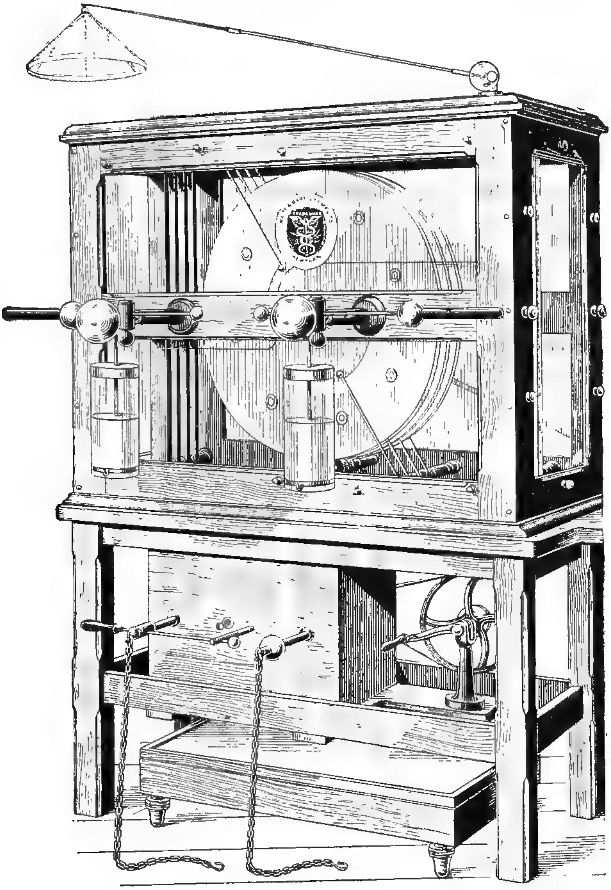


Toepler-Holtz electrical machine.

instead of plates were made to revolve between various appliances. In the latter part of the last century the influence or induction static machines were first used, but attracted little attention. In the franklinic machines now used the electric current is started by

friction, it is increased and continued by induction, and Leyden jars are employed to store or condense the electricity. The first important influence machine was made by Holtz, of Berlin, in 1865, and was known as Holtz's electrophanus machine. In the Holtz machine, as first made, one revolving and one stationary plate were used. The present improved apparatus is known as the Toepler-Holtz machine, and one form of it is shown in Fig. 188. The instrument,

FIG. 189.



The Ranney improved static machine, with charger attached.

of comparatively small size, has some therapeutic powers, although Morton asserts that curative results are obtained only from powerful machines and powerful administrations. An excellent larger form of static electrical machine is shown in Fig. 189. The main machine used in treating the patient is charged by a smaller one of what is known as the Wimshurst's pattern. Some other improvements of recent addition are the doing away with the catskin on the stationary

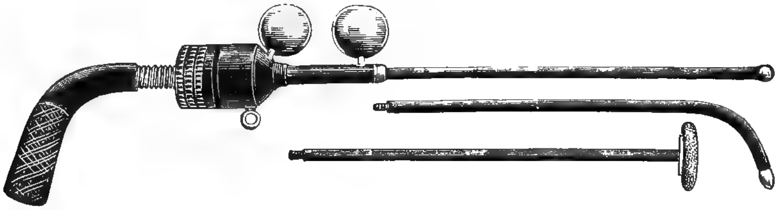
plates, and dispensing with the rubbers, which prevents wear of the lacquer on the revolving plates. The large machine can be charged without opening the case, an important matter in the summer months. The charging is done by connecting the two chains of the pole pieces of the larger machine and separating the sliding rods about half an inch. On revolving the small machine for a minute, and then starting the larger one, the latter will be found fully charged.

Static Electrodes and Accessory Appliances.—Various electrodes and other accessories are required for static applications. The chief of these are an insulated platform with a wooden stool or chair, brass chains and hooks for proper attachments, two or three pairs of Leyden jars of different sizes, with a brass rod to connect them, and a set of electrodes, the most important of which are brass balls and points, wood balls and points, rollers of metal and of wood, an umbrella electrode, and special electrodes for Morton's interrupted static current. Some pieces of catskin and a supply of calcium chloride will also be found useful, the latter to be placed within the case to absorb moisture.

Methods of Applying Static Electricity.—The chief methods of applying static electricity are by *insulation*, by *sparks*, by the *static breeze*, by the *aigrette*, and by the *static interrupted current*. In the method by insulation the patient is placed upon the insulated stool or platform and is connected with the machine by a chain. The other pole or conductor is then connected with the earth, or "grounded," as it is termed, which is often done by attaching it to a gas fixture or to the stopcock of a water basin. The conductors are drawn widely apart, and then the plates are revolved rapidly and the patient becomes charged, which may be shown by the rising of the hair, and even by the bluish or purplish light emitted from pointed portions of the body. The insulated stool or platform may be connected with either the positive or the negative pole, and the patient can thus be charged with positive or negative electricity of high potential. The methods of using the spark are *direct* and *indirect*. In the direct method the patient is put upon the insulated stool or platform, and this is connected with one of the conductors of the machine. The electrode or applicator is attached to a chain which is connected with the other conductor, and the rods joined to the two conductors are separated, so as to give a spark of greater or less length, and the applicator, held by the physician, is approached to the patient. The effect may be much increased by attaching small Leyden jars to the poles of the machine and then connecting their outer coating by a metallic rod or chain, bringing the poles or conductors into closer approximation. A severe shock can be given by this method, and it is necessary to be careful in its administration. In the indirect method of taking sparks, the patient being insulated and charged as described above, an electrode or applicator connected with a gas pipe

or faucet is approached to him, when sparks pass between the patient and the electrode. If, instead of a bulb or ball, a pointed electrode is used, a fan shaped spark or brush, the *static breeze*, will pass between the applicator and the patient. A so called electrostatic *current* is produced by passing the insulated ball electrode over the patient's clothing, which acts as an insulator. Ozone is developed during the action of an electrical machine, owing to the splitting by the electrical spark of molecules of oxygen into single atoms, some of which coalesce to form molecules of ozone. It can be administered to the patient by an insulated disk electrode furnished with points, which

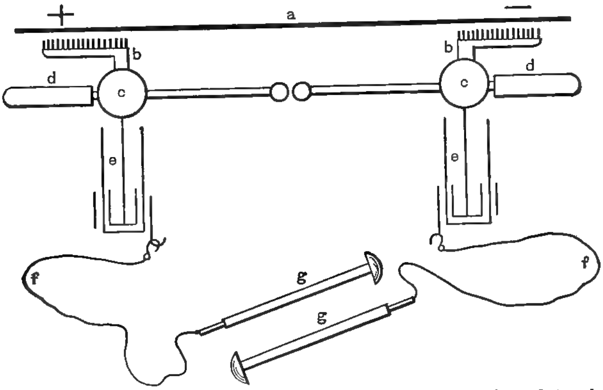
Fig. 190.



Morton's electrode for applying the franklinic interrupted current.

is held near the patient's open mouth, but not near enough to allow sparks to pass. The patient, breathing deeply at the time of the application, inhales the ozone. What is termed the *aigrette* is produced by bringing a rather blunt piece of wood or metal near the patient, which gives a form of discharge intermediate between the spark and the breeze. W. J. Morton was the first to describe a method of developing a rapidly interrupted and graduated current by means of a circuit breaking electrode (Fig. 190), two brass balls

Fig. 191.



Morton's method of applying the static induced current: a, rotating plate; b, b, collecting combs; c, c, prime conductors; d, d, discharging rods; e, e, Leyden jars; f, f, conducting cords; g, g, sponge or other electrodes.

constituting the circuit breaker. The spark circuit breaker practically represents the vibrator in the primary circuit of an induction

coil, the specific inductive capacity of the air replacing the spring and its magnetic attractability. In applying the interrupted static current (Fig. 191) the discharging rods of the machine are brought into contact; and if a Holtz machine is used, the connecting rods which unite the Leyden jars are removed, and to them are hooked conducting cords attached to the electrodes. The machine is set in motion and the discharging rods are separated very slightly. The current can be graduated to any strength by the amount of the separation of the poles and the size of the jars. It is not necessary to insulate the patient.

Nervous Diseases in which Static Electricity is Useful.—The nervous diseases in which static electricity is of genuine value can be summarized in a few words. They are (1) neurasthenia and hysteria; (2) chorea; (3) obstinate neuralgia or neuritis; and (4) some forms of local spasm, tremor, and paralysis. It has been strongly recommended by a few observers, as by Shaw and Morton, in the treatment of poliomyelitis. The good effects of static electricity are due chiefly to counterirritation, reflex inhibition, or psychic impression. Its method of action has been compared by Morton to the action of blisters, sinapisms, the cautery, acupuncture, hypodermatic injections of water, and to the effects of tuning forks, percuteurs, magnets, and metals. Early in the history of static electricity, excitators or applicators made of different substances were used in the treatment of various affections. It was held that catharsis was produced by thus carrying purgative substances into the system. Becquerel described the transport of ponderable substances by electric discharges. Proofs of the transport of substances by static electricity are that the odor differs with the excitation, that different therapeutic results are obtained with different excitations, and that loss of weight is sustained by metallic excitation if this is continued for some time.

Magnets in the Treatment of Nervous Affections.—Large magnets applied to the irritable spines of the hysterical, as shown by Benedikt, will sometimes cause the sensitiveness to disappear; and diverse effects, as the relief of hysterical contractures and of anesthesia, can be produced by the use of magnets. Peterson and Kennelly performed a number of physiological experiments with magnets of enormous power at the Edison laboratory, at Orange, New Jersey, and concluded that the human organism is in no way affected by the most powerful magnets known to modern science; that neither direct nor reversed magnetism exerts any perceptible influence upon the iron contained in the blood, upon the circulation, upon ciliary or protoplasmic movements, upon sensory or motor nerves, or upon the brain. The therapeutic effects achieved by the use of magnets would therefore seem to be mainly through mental impression.

GENERAL THERAPEUTICS.

General Hygiene.—Many diseases of the nervous system are directly or indirectly due to *heredity*, and it is therefore of the highest importance for their prevention that care should be taken in contracting marriages. The marriage of relatives, although not necessarily productive of evil, should as a rule be avoided. If the entire stock is healthy, no evil is to be apprehended, but if a tendency to any serious constitutional affection exists, it may be intensified in the offspring by intermarriage. Much can be done to prevent the development of nervous affections by the careful *training* and *education of children*, which should be individualized as far as possible, especially for children with neurotic tendencies. Education should be as objective as possible. *Sleeping* should be carefully regulated, but the number of hours required cannot be stated absolutely. The following may serve as a general guide : four years of age, twelve hours ; seven years, eleven hours ; nine years, ten and a half hours ; fourteen years, ten hours ; seventeen years, nine and a half hours ; twenty-one years, nine hours ; twenty-eight years, eight hours. *Alcohol* and *tobacco*, and even *tea* and *coffee*, are undoubtedly injurious to many neurotics. Travel, amusement, and natural exercise, skilfully directed, may be made more efficient in the treatment of the nervous and the insane than drugs or special therapeutic procedures.

Diet.—An abundance of *good food* is as necessary for brain workers as for muscle workers. Properly prepared nitrogenous food is necessary for brain and nerve work. Neurasthenics need easily assimilated food in small bulk but of high nutritive value. For them some of the artificial preparations, such as peptonoids, beef extracts, malt, and malted milk, are often of great service. Experience should guide as to their use. Often too little water is drunk both by nervous patients and by others. The suggestions given by Seguin with reference to diet can usually be followed with advantage. Concerning the diet and hygiene of nervous patients, he says that lithemia and oxaluria frequently accompany neurasthenia, and the excessive use of sweet and starchy foods is a potent cause of this condition. In regard to diet for nervous patients generally, they should drink large quantities of water, and, as they usually avoid a diet of fatty foods, physicians should insist on their eating pork, fat roast beef, and butter, and on their using cream and cod liver oil. Sweet and starchy foods should be eaten very sparingly, as they require to be transformed into food by complicated chemical actions within the body, while oil, butter, cream, and fat are already prepared for emulsion. All green foods of the spinach group are of special value.

Climate.—Under general etiology the effects of climate, and especially of high altitudes, have been considered. Notwithstand-

ing the injurious influence sometimes exerted by such altitudes, inland regions of moderate elevation may be of great service to anemic and debilitated nervous patients. Camp life is for them especially beneficial. Most forms of organic nervous disease do best at the seashore or at low inland levels. Some sleepless and neurasthenic patients are immediately and permanently benefited by the seashore. Now and then, however, cases of insomnia are not only not benefited but are made worse by a stay by the sea. Such neurasthenics, as advised by Daly, should be sent not too far inland, to get away from the ocean's roar and still have the tonic effects of the sea air. Idiosyncrasies and differences in the underlying causes of the insomnia doubtless have much to do with the varying reports as to the effects of the seashore in insomnia and other nervous disturbances. Patients whose pains are aggravated by the east winds or before storms may at first become worse at the seashore, but when they have become stronger they will often make marked improvement. (Boardman Reed.) For the sleeplessness of brain exhaustion sea air, with tonics, good food, massage, electricity, light exercise, and pleasant surroundings, will be found beneficial. A most important element in determining whether a change of climate will be beneficial to a patient is the mental attitude of the invalid. "If by removing the patient from his surroundings to others, a hopeful expectancy of good can be made to take the place of despairing apprehension, then a possibility of improvement is established which did not exist. But if the converse of this takes place, none of the natural advantages of climate can overcome the depressing influence of low spirits." (Ford.)

Hydrotherapy.—In continental Europe hydrotherapy has long held a place in the treatment of many nervous diseases, particularly in the "cures" or courses of treatment at various spas and health resorts, and in recent years it has been largely introduced into this country, but deserves to be more generally used. The treatment may be either local or general. For the thorough carrying out of hydrotherapeutic treatment a special room or rooms with waterproof walls and floors are necessary, although much can be done by improvised methods in a house with a good bath room. Appliances of various kinds are coming more and more into general use, as forms of apparatus for covering the limbs, and the hot box in which the whole body of the patient, with the exception of the head, can be placed. Some of the general laws of hydrotherapy are as follows: cold and warm baths affect the central nervous system in a reflex manner, stimulating the sensory nerves of the skin and the vasomotor nerves; short cold baths, especially when combined with sprinkling, showering, or rubbing, are powerfully stimulating, exhilarating, and tonic; prolonged warm baths and the hot pack are relaxing, fatiguing, and soporific; warm baths, by soothing

peripheral irritability, exert a calming influence over the central nervous system; cold applications to the skin stimulate the vaso-dilator nerves, dilate the peripheral vessels, and increase the blood pressure; warm applications also dilate superficial capillaries, but by diminishing the tone of the vessel walls reduce arterial tension. To lower the irritability of individual nerves, or of the entire nervous system, prolonged warm baths or the hot pack are indicated. (Peterson.)

Various Forms of Baths.—In the *cold plunge* the patient suddenly plunges or is immersed in cold water, staying only for a moment, and when he comes out is rubbed vigorously. *Brine baths*, either hot or cold, contain about twenty-five pounds of salt to thirty gallons of water. The *half bath* is taken by the patient reclining in a tub only partially covered by the water, while at the same time vigorous friction is performed, or water is poured or dashed over him. In the *sitz bath* the patient sits in a tub of water with the feet outside on a raised surface; the water should reach the umbilicus. *Mud baths* are prepared by mixing well seasoned earth containing mineral matters with water containing the same mineral matters. In treatment by the *dry pack* the patient is wrapped tightly in one or two blankets, which are surrounded by an outer cloth. In the *hot wet pack* one or two blankets are spread upon a bed, and on these is placed a large sheet which has been dipped in very hot water; this is wrapped close around the naked patient. *Turkish* and *Russian baths* are useful in some forms of nervous disease, but the condition of the heart, vessels, and lungs, and the general powers of resistance of the patient, should be considered. The most frequently used local *douches* are hot and cold, or the alternating hot and cold or *Scotch douches*. Jets of water can be thrown upon the spine from the nozzle of a tube connected with the water supply of the house, or with an apparatus made for this purpose, into which water is forced by compressed air. Similar results may be obtained by hot or cold affusion or by the use of a strong spraying apparatus.

Thermic Mineral Baths.—One of the best methods of treating multiple neuritis locally is by daily applications of very hot water, afterwards wrapping the portion treated in rubber dam, or in some similar material, to keep up warmth and gentle perspiration. Vogt has published the results of the treatment of forty cases of neuritis by thermal baths, nearly the entire number being cured or greatly improved. The baths were used in their natural temperature (32° C. or 90° F.) for from ten to fifteen minutes every other day. Thermal baths, such as are found at the German spas, and at the mineral springs in Virginia, North Carolina, Georgia, Arkansas, California, New Mexico, Colorado, Montana, and other parts of this country, are beneficial in the treatment of neuritis, especially when subacute or chronic. Peale has catalogued more

than six hundred mineral spring resorts within the limits of the United States, and nearly one hundred of these, mostly in the Western States and Territories, have facilities for thermic mineral baths. As thermal springs are found of all temperatures from warm to hot, any of the forms of ordinary warm and hot baths may be duplicated in them; but besides the effects following ordinary baths there are here others, resulting from the absorption of the mineral ingredients of the natural mineral waters. The external administration of natural thermal and mineral waters is frequently more advantageous than their internal use.

Temperatures of Different Forms of Baths.—In the following table are given the temperatures for the different forms of baths and the average duration of the baths, although these may be varied according to the intention of the treatment and the condition or idiosyncrasies of the patient:

Bath.	Temperature, Degrees Fahrenheit.	Duration of Bath.
Cold bath	60– 75	Five to ten minutes.
Cold brine bath	70	Five to ten minutes.
Cold jet and fan douche	50– 70	One minute.
Cold plunge	60– 70	Instantaneous.
Cold rain bath	50– 70	Five to ten seconds.
Cold wet pack	40– 60	One hour or more.
Half bath	65– 82	Ten to thirty minutes.
Sitz bath, cold	50– 70	Five to twenty minutes.
Sitz bath, tepid	70– 90	Five to twenty minutes.
Sitz bath, warm	90– 98	Five to twenty minutes.
Sitz bath, hot	100–125	Twenty to thirty minutes.
Tepid bath	85– 95	
Warm bath	90–104	Ten to thirty minutes.
Warm bath prolonged	70– 90	One half to two hours.
Mud bath	90–100	
Hot bath	104–110	
Hot wet pack	130–140	
Turkish bath	160–200	Fifteen to twenty minutes; for shorter time, followed by cold affusion.
Russian bath	Steam.	

Diseases in which Special Forms of Baths are Useful.—The following are some of the methods suggested for various nervous disorders: spinal douches or affusions for chorea; cold sitz baths for impotence, spermatorrhea, or incontinence; douches hot, cold, or alternating for spinal hyperesthesia; for migraine or anemic headaches hot compresses to the head; for congestive headaches hot foot baths; for melancholia douches gradually made cooler, also sometimes the shower or plunge bath; for the agitated form of melancholia general warm baths, with affusions to the head, or warm shower baths; for mania prolonged warm baths, with or without cold to the head, the hot wet pack, the cold wet pack; for neurasthenia

cold shower or immersion baths, douches, and affusions, at first cautiously used; mud baths for rheumatic and gouty neuritis; the hot wet pack for insomnia and excitement; Turkish and Russian baths for alcoholism, chronic neuritis and myelitis, and insomnia. *Surf sea bathing* is admirable for toning up debilitated nerves and muscles, and *cold brine* or *sea water baths* are also of value, and may be used when the patients cannot stand the buffeting of the waves. *Hot brine baths* are both sedative and stimulant in action. Among incurable diseases which may be ameliorated by hydrotherapy are locomotor ataxia and other forms of sclerosis, and chronic myelitis, with secondary degenerations. When hydrotherapy is used, strict attention should be paid to the organic conditions. In locomotor ataxia, and in general paralysis of the insane, for instance, it is important to proceed with caution. Vasomotor and trophic affections, like erythromelalgia, angioneurotic edema, and abnormal sweating, are sometimes amenable to hydrotherapeutic treatment.

Massage.—By the best authorities the essential massage procedures are included under four heads,—namely, *stroking*, *friction*, *kneading*, and *percussion*. Stroking is a light, continuous pressure, made by the under surface of the fingers or by the palm of the hand, the movement being usually from the periphery of a limb or

FIG. 192.

Friction of the fingers in massage treatment.

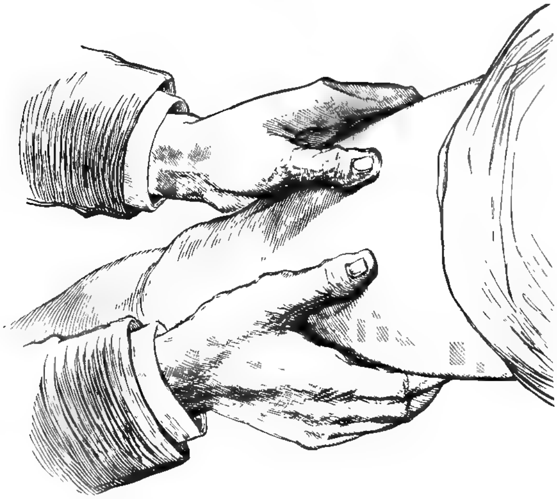
part towards the centre. Friction or rubbing is a forward movement of the hand with forcible pressure; and it is executed in various ways, according to the particular purpose to be accomplished. Kneading is a process of deep grasping and working of the tissues with the thumb, fingers, and thenar eminences. One or both hands

can be used. Percussion is striking or tapping with the hand or fingers, or with a hammer or other instrument. It can be performed with the ends of the half flexed fingers, with the flat of the fingers extended and separated, with the ulnar border of the hand, or even with the closed fist. In the lighter forms of percussion, which are most employed, the movement is from the wrist. These different methods are illustrated in Figs. 192, 193, 194. No one should attempt treatment by massage without training and preparation, and even with these a certain knack and capacity for the work are necessary in order that a masseur or masseuse may be successful. Twisting and mauling movements should be avoided. For many peripheral affections, such as palsies of the face or limbs, neuralgia or neuritis, local massage may be all that is required; but neurasthenia, hysteria, nervous

Fig. 193.

Position of the hands in friction of broad surfaces.

Fig. 194.



Position of the hands in compression or deep massage.

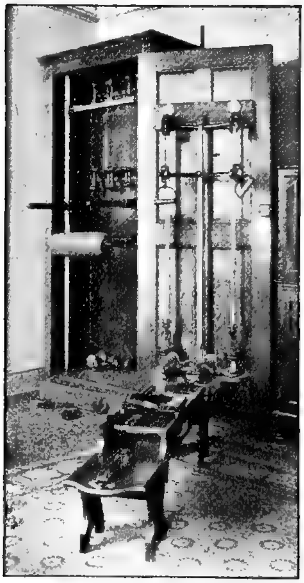
dyspepsia, and other neuroses call for general massage. Massage of the head and neck is often an admirable adjuvant treatment of headache and insomnia.

Movement Treatment.—Movements are *active*, *passive*, *single*, and *duplicated*. Active movements are those which are under the control of the one making or taking part in them. Passive movements come from without; they are performed on the patient and independently of his will. He is subjected to pushings and pullings, to flexions and extensions, to swingings and rotations. A single movement is one in which only a single individual is engaged. In duplicated active movements the elements of resistance play an important part. The operator, with carefully considered exertion, performs a movement which the patient resists, or the reverse. By changing the manner of operating from time to time any group of muscles can be brought into play. Movement apparatus run by steam is sometimes used, and under careful supervision may be of great benefit. To some extent semiactive and semipassive or duplicated active movements may be carried out by such machinery, which may be made to cover the whole range of passive exercises.

Systematized Active Exercises.—The methods of systematized active exercises chiefly used by me are (1) the respiratory exercises of Blaikie, with or without dumb bells; (2) exercises with pulley and weight apparatus. The Pichery system of elastic straps, styled "opposition gymnastics," can also be used. Elastic cords with handles attached are firmly fastened in convenient places, and by making traction in different directions almost any muscular action can be elicited; but the force used cannot be as thoroughly controlled as by the pulley and weight apparatus. In the prolonged treatment of patients, some advantages accrue from the use, conjointly or alternately, of exercises with and without apparatus. Exercise treatment should be as far as possible personally directed, and the director of these exercises should be discreet and thoroughly well fitted for his work. The treatment should begin with the simplest forms of exercises, and these should be constantly increased and elaborated as the patient gains in strength and skill. Usually respiratory exercises should be combined with muscular movements, as on these two powers depends the ability to perform all bodily exercises. Inherent nervous force has also something to do with the capacity to perform bodily exercise. The special efforts of breathing include taking deep, full breaths through the nose and mouth, forced expiration as well as inspiration, counting with a loud voice while holding the breath, etc. The development of the lungs and abdominal walls, and the greater aeration of the blood which is conveyed to weak spinal or encephalic centres, make them of decided value in cases in which active movements are applicable. In my own practice I have used systematized active exercises in the treat-

ment of idiocy, insanity, chorea, hysteria, neurasthenia, nervous palpitation, lithemia, diabetes, curvatures, ataxia, chronic neuritis, muscular atrophy, and occupation neuroses. For gout and lithemia, to promote excretion and nutrition ; for anemia and spanemia, to assist assimilation and further oxidation ; for headache, sleeplessness, and nervous irritability, to soothe and calm the nervous system ; to aid elimination in cases of lead, arsenic, mercury, and other metallic poisonings ; and for diabetes, to favor the action of the skin and increase combustion, these exercises have a value which cannot be too highly extolled. In hysteria the advantages of any treatment which involves specific direction and the adroit calling out of the volition of a patient must be evident. Even in paralysis from organic disease of the brain, as in many cases the amount of palsy is disproportionate to the cerebral lesion by which it has been initiated, efforts consistent with safety should be made to unite again the paralyzed limb with the volitional centres. Special gymnastic methods employed in the treatment of chorea will be given when that disease is considered. The treatment by rest, seclusion, massage, and electricity can be successfully combined with that by systematized exercises. After the patients have progressed to a certain extent and their nutrition has been placed on a firm basis, respiratory exercises without apparatus, or with light dumb bells, should be carefully begun. In Fig. 195 is shown a cabinet gymnasium, made for the author by A. G. Spalding & Bros., of Philadelphia. It contains almost everything necessary in the treatment of office or home patients by systematized gymnastics. In the treatment of ataxic affections the use of balancing or acrobatic gymnastics is of some value ; and Mortimer Granville advises a method which consists in directing the patient to stand with his eyes closed in his bath, after pouring a small can of water down his spine, or applying a mustard poultice over the full length of the spine for ten minutes or a quarter of an hour. Even bicycling has been recommended for nervous patients by Graeme Hammond and others. Cases of neurasthenia, melancholia, and other nervous troubles will derive much benefit from our present hobby, for the wheelman must develop—whether he chooses or not—his will, his independence, his self-reliance, and the accurate control of his muscles. (Egbert.)

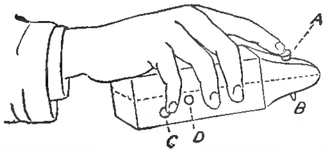
FIG. 195.



Cabinet gymnasium.

Vibratory Therapeutics.—Mortimer Granville, Boudet, Charcot, and others have strongly advocated the treatment of pain by mechanical vibrations. Granville has devised several forms of percuteurs for exciting the nerves by vibration. One of these is a clockwork instrument (Fig. 196). The others are worked by electricity, and are more convenient and have a wider range of usefulness (Figs. 197, 198). Various accessory appliances, as disks, brushes and points similar in construction to electrodes, are used for percussing and vibrating different regions and tissues. The method of treatment with the flat disk is shown in Fig. 197, and with the wire brush in Fig. 198. Light or firm pressure can be used according to the depth of the affection and the tenderness of the surface. Headaches are often treated by percussion with the disk, and superficial pain, various paresthesias and dysesthesias, by application of the wire brush to the skin. Charcot, having observed that patients suffering from paralysis agitans were relieved by railway or other long journeys, had a vibrating armchair constructed to give a movement similar to that of a running train. A vibrating helmet has been invented by Gilles de la Tourette, which can be run by a small motor. These forms of vibratory treatment sometimes relieve symptoms, and in peripheral nerve affections are occasionally curative. Whether they are as

Fig. 196.

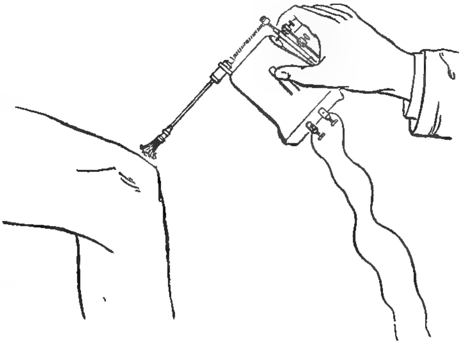


Mortimer Granville's clockwork percuteur, showing the method of using it: *D*, winding pivot; *C*, ivory button; while this is pressed by the finger the hammer continues in action; when the pressure is taken off it stops; by pressing the button *A* the length of stroke can be increased, and the speed slightly reduced, while the force of the blow is augmented; *B*, the pointed ivory hammer with which the percussion is made; a flat headed hammer, or a brush, can be substituted for the ivory point.

Fig. 197.

Mortimer Granville's flat disk electrical percuteur.

Fig. 198.



Mortimer Granville's wire brush electrical percuteur.

efficient as alternating electrical currents of great rapidity may be questioned.

The Weir Mitchell Rest Treatment.—Rest in the treatment of nervous disease, first reduced to a system by Weir Mitchell, has become an acknowledged therapeutic measure in all parts of the world. It includes rest in bed, seclusion, massage and Swedish movements, electricity, and systematized feeding. For full details of this treatment the writings of Mitchell, Playfair, Sinkler, and others can be consulted, and with reference to massage and electricity the instructions given elsewhere in this chapter are sufficient. The massage treatment and the electrical treatment are usually general. Patients should be carefully chosen, for any one who applies the rest treatment at haphazard will score more failures than successes. The rest treatment is most valuable in neurasthenia and hysteria, but it may be used with advantage in other affections, as chorea, exophthalmic goitre, neuritis, and myelitis, in some cases of melancholia and mania, and even in locomotor ataxia for the relief of pain and to improve the general nutrition. The opium, chloral, alcohol, or other drug habit can also sometimes be advantageously treated by this method. The moral influence of the physician is of great importance in the rest treatment. The patient must be made to understand thoroughly what the treatment is to be before it is begun, and it is better for the doctor not to undertake it until he has the full consent of the patient and that of responsible relatives or friends. A physician who is fitted to succeed in carrying out a treatment of this kind may find occasion to change his programme, but it is a serious mistake to modify too readily a plan which has been adopted.

Full and Partial Rest Treatment.—The treatment may be either *full* or *partial*. In the former the patient is kept absolutely at rest, and is waited on hand and foot for periods varying from one month to two. Writing, reading, crochetting, etc., are absolutely forbidden, but the patient is read to or talked to by the nurse as seems best. After a variable period the patient is allowed to sit up for a short time once a day, and soon this period is lengthened and the number of times a day increased. Systematized active exercises, muscular and respiratory, may now be used, and can be gradually increased; later the patient is permitted to walk or drive. In the partial rest treatment the patient is kept at rest for stated periods during the day. Seclusion is usually of paramount importance. In Philadelphia, houses for private patients are generally used. Nurses, healthy and tactful, and well adapted to be companions as well as to serve their patients in ordinary nursing capacities, are necessary. The best nurses for these cases are young, active, quick witted, intelligent women, capable of firmly but gently controlling the patient. (Mitchell.)

Diet in the Rest Treatment.—An essential feature of the rest treatment is a combination with it of excessive feeding, made possible by passive exercises obtained through the steady use of massage and electricity. In most cases the diet should at first be chiefly skimmed milk, carefully kept fresh, which, at the start, is best given every two or three hours in quantities of about four ounces, taken slowly with a spoon or sipped. Horlick's malted milk or some of the infant foods may be advantageously alternated with the milk; and after a week or ten days some of the best extracts of beef, or home made beef tea, may be used. Soon broiled meats, oysters, chicken, and vegetables sparingly, are added in increasing amount. Properly administered massage and Swedish movements and the use of electricity will do much to counteract the tendency to constipation which is not unusual, but it is occasionally necessary to use drugs, among the best of which is the pill of strychnine, aloin, and belladonna, either with or without cascara or podophyllin. Draughts of hot water taken before breakfast will often prove of service. Other simple remedies are liquorice powder, tamar indien, or pills of ox gall and watery extract of aloes. Every second or third day it may be necessary to unload the lower bowel with a large hot water enema. Each patient should be carefully studied as to the effects of milk diet. Morton has devised a tumbler which is convenient for giving milk by accurate dosage. It has engraved upon it the hours when the milk is to be taken, and also the quantity to which it is to be increased from day by day.

SCHEDULE FOR FULL REST TREATMENT.—(J. K. Mitchell.)

Cocoa at 7 A.M.
Cool sponge bath with rough rub, and toilet for the day.
Breakfast at 8 A.M., with milk.
Rest an hour after.
Eight ounces peptonized milk at 10 A.M.
Massage at 11 A.M.
Eight ounces milk or soup at 12 M.
Reading aloud by nurse half an hour.
Dinner at 1.30 P.M.
Rest an hour.
Eight ounces peptonized milk at 3.30.
Electricity at 4 P.M.
Supper at 6.30, with milk.
Rest an hour.
Reading aloud by nurse half an hour, 8 P.M.
Light rubbing by nurse with drip sheet at 9 P.M.
Three ounces malt extract with meals; tonic after meals.
Eight ounces peptonized milk with biscuit at bedtime, and a glass of milk during the night, if desired.

Laxative (cascara), 10–30 drops p. r. n.

Later, Swedish movements are added after the massage.

SCHEDULE FOR PARTIAL REST TREATMENT.—(S. Weir Mitchell.)

A.M.—On waking, cup of cocoa or beef extract.

Take bath. (Temperature given.) Lie down on lounge while using drying towels; or, better, be sponged and dried by attendant. In this process the surface to be rubbed red, or, if drying one's self, flesh brush to be used.

Bed or lounge again.

Breakfast.

Before each meal take three ounces of malt extract; aperient at need in malt.

Tonic after each meal.

Detail as to breakfast diet.

If eyes are good, may then read, seated in bed.

At 10 to 11 A.M. one hour's massage.

Rest one hour; may be read to, or read if eyes are good, or knit. At this time, 11 A.M., four ounces of beef soup or eight ounces of milk.

At noon may rise, dress slowly, resting once or twice a few minutes, and remain up until 3 P.M. (See children, attend to household business; see one visitor, if desirable.)

1 to 1.30 P.M.—Malt, etc., and lunch. Detail as to diet. At first, as a rule, let this meal represent dinner.

Tonic, and after it rest on lounge, occupied as above, reading or being read to.

If possible, drive out, or use tramway, so as to get air. *Walk as little as possible.*

On return from drive repeat milk and soup.

About 5 P.M., electricity, if used at all.

Rest until 7.

Supper at 7. Detail as to meal. Malt as before, with or without aperient, as occasion demands.

Tonic.

To spend evening with family as usual. Best not to use eyes at night for near view.

Bed at 10 P.M.

No letters to be written for two months, when most of these details have to be revised.

Suspension Treatment.—Suspension, a treatment which has long been employed in spinal caries, a few years ago came into use in the treatment of locomotor ataxia, and since then has been largely employed in this and other degenerative and neuraxial inflammatory diseases. While little can be said in favor of its cura-

tive virtues in these affections, it certainly has been beneficial in relieving serious symptoms, and apparently it retards the progress of the disease. Motchoukowski, of Odessa, was the first to discover the therapeutic value of suspension, and published his plan in 1883. Charcot tested the method in 1888 with unexpected and striking results in the treatment of locomotor ataxia. Gilles de la Tourette, Eulenberg, Mendel, Blocq, de Watteville, Weir Mitchell, D. D. Stewart, and many others, have applied the treatment to ataxics with satisfactory results. The forms of apparatus are all modifications of Sayre's suspension apparatus for the application of plaster jackets. In that shown in Fig. 199 the straps are made to go under the armpits and around the head and neck. Weir Mitchell, in order to take pressure from the armpits, modified the apparatus usually employed, as shown in Fig. 201, in which the lift is made from the elbows and the head is drawn up as may be necessary. A separate pulley and cord are used to govern the amount of pull on the head. The vertical straps are widened under the elbows, and a wide band is carried around the front and upper arm.

FIG. 199.

The Charcot-Motchoukowski suspension apparatus.

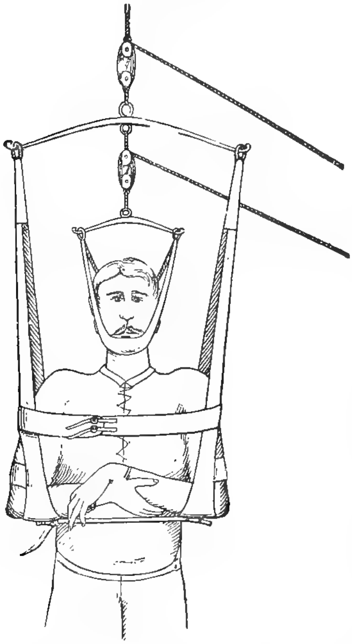
Method of Stretching the Spine.—The *Bonuzzi method* of stretching the spine, named after its deviser, requires no apparatus, and gives results comparable to those of suspension. In consequence of the result of some experiments made on the cadaver, this method is asserted to stretch the spinal cord more than three times as much as suspension, and its use is strongly recommended by Benedikt, Bernhardt,

FIG. 200.

Application of the chin neck band of the Charcot-Motchoukowski suspension apparatus.

and others. It is certainly efficient in locomotor ataxia in improving the station and gait and in alleviating the pain. It is necessary to be somewhat cautious in its use, as local spinal and muscular traumatism may occur. The patient being laid on a couch, with the head and shoulders raised by a pillow, the operator grasps the ankles and carries the feet and limbs upward and flexes the trunk until the knees reach the forehead and even the shoulders, the diverging ankles being then pulled downward towards the floor. Stretching the legs in the treatment of sciatica is carried out by a similar method. A method of suspension recommended by Bogroff, of Odessa, is to place the patient on an inclined plane at an angle of 60 to 100 degrees for ten to fifteen minutes, only straps for the arms being used. Stillman has suggested an apparatus by which the cord and the spinal column can be extended while the patient is in a horizontal position or hanging from the neck with the back resting upon a curved board.

FIG. 201.



Mitchell's suspension apparatus.

Surgical Treatment of Nervous Affections.—The surgical treatment of nervous affections will be considered under special diseases. It includes nerve stretching, section, resection, suturing, and ligation; trephining of the spine and skull for tumors, abscesses, hemorrhages, etc.; paracentesis of the ventricles and of both the cranial and the spinal dura.

Psychic Therapeutics.—For nervous patients, the psychic or mental treatment, or "moral treatment," as it is sometimes termed, is often of first importance. In affections like neurasthenia, hysteria, and the traumatic neuroses the personal influence of the doctor may be the potent agent in effecting a cure; in organic incurable diseases of long duration it supports the patient in his sufferings and encourages him to a useful life in spite of them; in some types of insanity, like mania and melancholia, it checks, guides, and encourages the patient. The neurologist must be authoritative and yet not domineering, firm and yet not harsh. He must be unflinching to withstand patients and their friends when he has a certain course to follow. Suggestion without hypnotic procedure may lead the way to cure, of which innumerable instances are given in works like Tuke's "Influence of the Mind upon the Body."

Hypnotism.—In recent years hypnotism has been turned to many therapeutic uses, but with only moderate success. The consensus of conservative opinion is against its value in the treatment of insanity; it is of less value than ordinary suggestion. Temporary success has been obtained with it in the treatment of inebriety. Its most useful field is probably in the treatment of hysterical affections, and even here it may be useless or even worse; but this subject will receive fuller consideration elsewhere.

Local Remedies.—*Camphorated chloral* is a thick, syrupy liquid which results from rubbing together equal parts of camphor and chloral. Morphine, atropine, or chloroform may be added to it. For the relief of local pain it is painted over the surface with a brush or cotton, and may be kept in position a short time covered with oiled silk, but usually it cannot long be borne, owing to the burning pain produced. *Camphorated menthol*, composed of equal parts of menthol and gum camphor, combined with lanolin in the proportion of one part to four, may be used in the same manner and for the same ends as camphorated chloral, but it is not so efficient as the latter. The menthol pencils have had considerable popularity, but are of little value except where the pain is superficial and not severe. *Antifebrin*, for external use, can be combined with lanolin or vaseline in the proportion of twenty grains to the ounce. Among valuable anodyne ointments are those of veratrine, aconitine, atropine or belladonna, and opium. Liniments are not entirely out of date, but only the stronger preparations, such as the compound chloroform liniment, need to be considered.

Mercurial Inunction.—When mercurial inunction is used for nervous syphilis, as for other purposes, the physical condition of the patient must be attended to most carefully. The best preparation is the official ointment of mercury of fifty per cent. strength, which can be made with lanolin. Oleate of mercury may also be employed. One fourth to one half drachm daily is a safe amount to use in ordinary cases, and this may be increased to a drachm where a rapid effect is desired and where the patient stands the treatment well. An exact amount can be administered by having the prescription divided into fifteen or thirty grain doses, each wrapped in oiled or paraffin paper. General or local baths should be used before its administration, and whenever possible it is well to have a trained masseur or masseuse make the application.

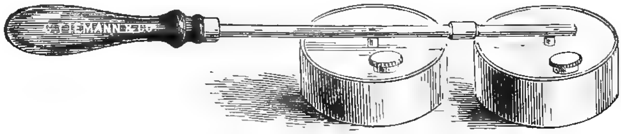
Cold Applications.—Cold is of great value in the treatment of neuralgia, neuritis, and facial and other forms of local spasm, and can be administered by ice bags, by packing the limb in ice, or by various sprays. Rhigolene jets or the ether spray may be used. The eyes should be protected when the application is made to the face, and care must be taken not to use the spray too long or too many days in succession, or a slough may be produced. Mixtures of salt and ice

furnish a simple and convenient method of applying intense cold. Bags filled with ice, or ice water circulating through bags, may be employed, and different saline solutions may be mixed with the ice in order to secure definite temperatures. In 1884, Debove recommended chloride of methyl as a freezing agent for sciatica, facial neuralgia, and other painful affections, and Jacoby introduced this method of treatment to the American profession in 1887, recommending as the two best refrigerants chloride of methyl and fluid carbonic acid. Dobisch recommends for the purpose of procuring local anesthesia a spray composed of ten parts chloroform, fifteen parts sulphuric ether, and one part menthol, to be used with a spraying apparatus. After one minute's application of this spray complete anesthesia of the skin and neighboring tissues is obtained, which lasts from two to six minutes and suffices for the performance of minor operations, and may be also used cautiously for local painful affections. In one of the forms of spinal ice bag the ice is applied in a bag that reaches from the lower part of the neck to the sacrum. The bag should have two or more compartments, and the ice should be in small pieces. It should be worn for one or two hours daily, or, in acute disease, until the disturbance is subdued.

Hot Applications.—The hot spinal bags should not be applied at a higher temperature than 120° F. Hot water bags, which can now be had of different patterns, are useful and easy of application for the treatment or temporary relief of many nervous affections accompanied by pain. One of the most soothing and effective methods of treatment of either localized, diffused, or multiple neuritis is by means of hot douches or hot sponging of the affected parts two or three times daily, in the intervals wrapping the limbs or neuritic areas in some material, such as rubber dam, which will keep the parts warm and induce a gentle perspiration. In continuing the use of either hot water or ice, strict attention should be given to the effects, and neither should be continued when uncomfortable or painful results ensue.

The Revulsor.—Hamilton has described an instrument for the alternate application of dry heat and dry cold, which consists of

Fig. 202.



Hamilton's hot and cold revulsor.

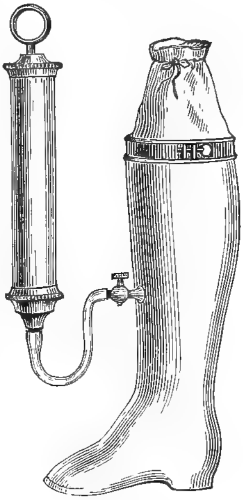
two chambers of brass, three inches in diameter by one and a half inches in depth. These have screw plugs inserted, so that they may be removed and the chambers filled, one with salt and cold water and the other with hot water. These chambers are fixed on

a rod and separated by an insulating or nonconducting substance. The rod terminates in a handle. The flat surface, covered by thin flannel, is placed against the bare back, on either side of the spinous processes of the vertebræ, and the instrument is moved up and down quite rapidly. As the heated surface moves instantaneously to where the cold one was before, the effect is quite marked.

Lavage.—Lavage or washing out of the stomach, which has become so popular for many gastric disturbances in recent years, is sometimes useful in the dyspepsias which accompany neurasthenia and for the relief of sitophobia, or refusal to take food, among the insane. Régis recommended it for the latter purpose in 1880, and also for the treatment of melancholia itself. The exact composition of the gastric juice should be determined in order to decide as to the liquid to be used.

Cupping Apparatus.—Various forms of cupping apparatus—from the small cup for the temple to the Junod's boot (Fig. 203), made to encase an entire limb—are occasionally employed. The application of revulsives to the lower extremities rather than to the spine is called for in some acute and chronic diseases of the spinal cord.

Fig. 203.



Junod's boot.

Thyreoid Treatment.—Brown-Séquard, in 1856, recommended the use of various glands of the human body for therapeutic purposes; and again, after Schiff, in 1880, demonstrated that ablation of the thyreoid gland resulted in artificial myxedema, Brown-Séquard suggested the injection of the extract of thyreoid for the cure of this disease. Fenwick, Murray, and Wallace Beatty were among the first to adopt this recommendation. Horsley called attention to Schiff's observation on the transportation of the thyreoid gland into the peritoneal cavity, which showed that thyreoidectomy lost its dangers and an essential amount of its effects, and suggested the thyreoid gland of the sheep in cretinism and cachexia strumipriva. Vassale demonstrated the important physiological effects of intravenous injections of thyreoid juice in dogs from which the gland had been removed, holding that the thyreoid gland prevented autointoxication. Preparations of thyreoid are usually made from the thyreoid glands of sheep, and are of several kinds, as (1) the thyreoid, prepared by cooking; (2) thyreoid extracts, usually prepared by maceration with glycerin or alcohol; and (3) powders prepared by evaporation and other measures. The preparations are given either by the mouth or by hypodermatic injec-

tions. The dose usually administered is equivalent to one lobe or half a thyreoid. To obviate nauseous taste, the powders may be given in capsules or in keratine coated pills. About fifteen grains of the powder, as usually prepared, or thirty drops of the glycerin extract, represent about half an average thyreoid. It is believed that glycerin extracts will retain their virtues for eight or ten weeks, but the best results cannot be expected from a liquid preparation of a much greater age than this.

Thymus Treatment.—Extracts derived from the thymus gland have been used in progressive muscular atrophy, but no results of value have yet been reported. (Macalester and Dana.) Dercum has suggested that as the experiments of Brieger, Kitasato, and Wassermann show that the tetanus bacillus, although it grew in thymus infusion, did not develop spores, and that animals inoculated with such cultures were made highly immune to the cultures of tetanus grown in other media, the thymus juice should be tested with a view to a possible therapeutic effect; as its administration beneath the skin in cases of tetanus could certainly do no harm, and might do good, and it might also be administered by the mouth, like the thyreoid gland in myxedema.

Cerebrin and other Tissue Extracts.—Hammond has recommended the use of an organic extract made from the brain of an ox, and called cerebrin, which is administered hypodermatically in doses of five minims, diluted at the time with a similar quantity of distilled water. Various contributions on the subject have been published since the original paper of Hammond, some with approval, with reports of successful cases, many others critical and condemnatory. The subject may be considered unsettled, with the weight of authority against it. Among the diseases and symptoms for which it has been tried are neurasthenia, hysteria, neuralgia, melancholia, dementia paralytica, locomotor ataxia, and epilepsy. Extracts of other tissues than the brain have been largely used, as *medullin*, prepared from the spinal cord, *pancreatin*, from the pancreas, *gastrin*, from the stomach, and *cardin*, from the heart. Some of the results obtained are scarcely worthy of discussion. In all injections of animal substances the greatest care should be taken to have the procedure aseptic and antiseptic.

Testicular Therapy.—In 1889, Brown-Séquard put forth a remarkable claim as to the virtues of testicular fluid which attracted worldwide attention and almost equally wide ridicule and condemnation. These injections have been used in widely different diseases, largely those which involve exhaustion or degeneration of the nervous system, as neurasthenia, impotence, locomotor ataxia, brain exhaustion, melancholia, and other forms of insanity, and numerous atrophic and dystrophic diseases. The preparations are administered either subcutaneously or intravenously. They include

(1) the fresh testicular fluid, and (2) extracts obtained from the gland. The following are Brown-Séquard's directions for the preparation of the fluid used by him. To obtain the testicular fluid, the testicle is crushed in distilled water immediately after it has been taken from a dog or a guinea-pig. The crushing is always done after the addition of the water, which is in a quantity never exceeding three or four times the volume of the testicle. The liquid is then filtered through filter paper or a Pasteur filter. For each hypodermatic injection use nearly sixteen minims of the filtered liquid. The conclusions reached by Venturi and Frondo with reference to the injection of testicular remedies in the therapy of mental disease probably approach the truth. They conclude that such injections have given negative or at best only transitory results; some patients were rendered suddenly more excitable, and the spinal functions were reinforced briefly.

Immunity.—Immunity is that condition of the body which enables it to resist infectious or other morbid processes. The experiments of the last few years indicate its great value in the prevention of such affections as tetanus and hydrophobia; and doubtless in time other nervous and mental diseases of infectious origin may be favorably influenced or cured by immunization. Studies made by Behring have shown a uniform peculiarity of blood serum in one who has been rendered artificially immune, and this has been recorded formally in what is now termed Behring's law, which is as follows: "If an individual is rendered artificially immune against a certain infectious disease, then his blood or its serum has acquired the property of transmitting immunity against the same infectious disease to a susceptible individual (no matter of what species) into whose organism it can be brought in sufficient quantities." On this law is based the action of blood serum, or of the precipitates derived from it, in the treatment as well as in the prevention of tetanus and other infectious nervous diseases.

Serum Therapy.—As experiments have demonstrated that pathogenic microorganisms in the body will elaborate toxins which cause certain symptoms and diseases, and as the body cells or fluid constituents of such animals, suitably stimulated, are capable of elaborating antidotal antitoxins, it follows that the serum containing such toxins and antitoxins is a therapeutically active substance, which may improve or cure the disease, or produce immunity from its future visitations. (Threlkeld-Edwards.) A practical objection to the use of serum as a therapeutic measure is the large amount which is required for injection. The healing value of the serum is one thousand to two thousand times less than its immunizing value. Sixty-six grammes of immunized serum from the blood of the horse, containing 0.5 per cent. of carbolic acid, were injected into four places beneath the skin of the back in an

adult man suffering from tetanus; on the day following fifty grammes were injected, and still later forty-five grammes more. Tizzoni and Cattani obtained an alcoholic precipitate from the serum—a tetanus antitoxin—which they believed to have the same value as the serum itself. Tizzoni estimated that of his serum seventy cubic centimetres would be a dose for a case of moderate severity in man in the beginning. At a later stage two hundred and ten cubic centimetres would be required. This would equal five to six centigrammes of the alcoholic precipitate in the first instance and ten to twelve grammes in the latter. Dercum collected the records of thirty-four cases of tetanus of various origins treated either by the powder of Tizzoni or by the immunized serum. Of these twenty were successful. The first case of tetanus successfully treated with tetanus antitoxin was reported by Dr. Rudolf Schwarz, assistant at the surgical clinic at Padua. Tetanus antitoxin was sent by Professor Tizzoni, who obtained it from the blood serum of a dog which had been rendered strongly immune against tetanus. The antitoxin was used hypodermatically in doses of fifteen grains dissolved in water.

Nuclein Therapy.—Vaughan believes that the immunizing substance is the nuclein of the cell, that part which gives to the germ its distinctive properties. An animal may be rendered immune by treating it with the blood serum of another immune animal; but even in this case the immunity is primarily due to the antitoxin generated in the cells of the immune animal. A horse rendered immune to tetanus has the serum of its blood injected into a mouse, and this animal becomes for the time being immune to tetanus poison. It is supposed that immunity depends on the altered activity of the cells of the spleen, the bone marrow, the thyreoid and thymus glands, and possibly of other glandular organs, removal of these organs leading to the formation in the system of toxic substances. Vaughan's experiments with yeast nuclein showed that solutions of it rapidly inhibited the multiplication of the staphylococcus pyogenes aureus. Testicular nuclein from the testicles of a bull, a guinea-pig, and a rabbit acted in a similar manner. Five to twenty drops of a glycerin solution used hypodermatically in a case of nervous exhaustion had a marked stimulant effect. Vaughan also obtained nucleins from the thyreoid, and from the brain and spleen. He believes that the germicidal properties of blood serum are due to soluble nucleins, and that the germicides which hold most therapeutic hope must be of cellular origin. The word nuclein is used in a broad sense to indicate that part of the cell which under normal conditions is endowed with the capabilities of growth and reproduction. In nuclein therapy, as Vaughan suggests, the active substance can probably be used in larger doses than in blood serum therapy. The effect in immunity and cure being proportional to quantity, this must always be considered.

*Doses of some of the More Potent and Newer Drugs used in Neurological Practice.**

Acetanilid (antifebrin)	grs. 3–10	gm. 0.200 — 0.666
Aconitine	gr. $\frac{1}{400}$–$\frac{1}{100}$	gm. 0.00016 — 0.0006
Agathin	grs. 8–10	gm. 0.533 — 0.666
Amyl nitrite	m. 1–5	c.c. 0.066 — 0.333
Amylene hydrate	m. 30–60	c.c. 2.000 — 4.000
Antipyrin	grs. 5–20	gm. 0.333 — 1.333
Apomorphine hydrochlorate	gr. $\frac{1}{8}$–$\frac{1}{4}$	gm. 0.008 — 0.016
Bromamide	grs. 10–15	gm. 0.666 — 1.000
Butyl chloral hydrate (croton chloral)	grs. 5–10	gm. 0.333 — 0.666
Caffeine citrate	grs. 2–5	gm. 0.133 — 0.333
Chloralamid	grs. 10–30	gm. 0.666 — 2.000
Chloralose	grs. 3–10	gm. 0.200 — 0.666
Chloral hydrate	grs. 3–20	gm. 0.200 — 1.333
Cocaine hydrochlorate	grs. $\frac{1}{4}$–2	gm. 0.016 — 0.133
Codeine sulphate	grs. $\frac{1}{2}$–2	gm. 0.033 — 0.133
Coniine hydrobromate	gr. $\frac{1}{20}$–$\frac{1}{10}$	gm. 0.003 — 0.006
Curarine sulphate	gr. $\frac{1}{100}$–$\frac{1}{40}$	gm. 0.0006 — 0.0016
Duboisine sulphate	gr. $\frac{1}{120}$–$\frac{1}{60}$	gm. 0.0005 — 0.001
Eserine sulphate	gr. $\frac{1}{64}$–$\frac{1}{20}$	gm. 0.001 — 0.003
Exalgin	grs. $\frac{1}{2}$–5	gm. 0.033 — 0.333
Fluid extract of gelsemium	m. 3–10	c.c. 0.200 — 0.666
Hyoscine hydrobromate	gr. $\frac{1}{120}$–$\frac{1}{60}$	gm. 0.0005 — 0.001
Hypnal	grs. 10–20	gm. 0.666 — 1.333
Methacetin	grs. 5–15	gm. 0.333 — 1.000
Methylal	m. 10–30	c.c. 0.666 — 2.000
Nitroglycerin (spiritus glonoini)	m. $\frac{1}{2}$–3 †	c.c. 0.033 — 0.200
Paraldehyde	m. 30–60	c.c. 2.000 — 4.000
Phenacetin	grs. 2–10	gm. 0.133 — 0.666
Phenocoll hydrochloride	grs. 5–15	gm. 0.333 — 1.000
Salicylamid	grs. 3–5	gm. 0.200 — 0.333
Salipyrin	grs. 15	gm. 1.000
Salol	grs. 5–15	gm. 0.333 — 1.000
Salophen	grs. 15	gm. 1.000
Sulphonal	grs. 10–30	gm. 0.666 — 2.000
Tetronal	grs. 10–30	gm. 0.666 — 2.000
Theine	grs. $\frac{1}{2}$–2	gm. 0.033 — 0.133
Thymacetin	grs. 5–15	gm. 0.333 — 1.000
Trional	grs. 10–30	gm. 0.666 — 2.000

* To Dr. E. C. Seguin, of New York, the profession is greatly indebted for a series of valuable studies in the medicinal therapeutics of nervous diseases. The contributions of this distinguished neurologist, of which I have made frequent use, include papers first published in the *New York Medical Record*, *New York Medical Journal*, *Archives of Medicine*, *Journal of Nervous and Mental Diseases*, and other medical journals, most of them having been subsequently brought together in his "Opera Minora." One of the most valuable is a paper on "The Efficient Dosage of Certain Remedies used in the Treatment of Nervous Diseases," read before the Medical Society of the State of New York in 1882; another is entitled "Lectures on Some Points in the Treatment and Management of Neuroses," which appeared in the *New York Medical Record* during 1890, and was reprinted.

† Of one per cent. solution.

Efficient Dosage.—While drugs should not be abused, on the other hand they should be used in an efficient manner. Less harm is done by the large doses than by the reckless, long continued, or irregular administration of remedies. Uncured nervous diseases, such as choreas, cerebral and spinal syphilis, and certain neuralgias, are frequently cases in which the attending physician has prescribed the proper remedy but has exhibited it in doses wholly insufficient. The practitioner should be familiar with the maximal doses of physiologically active remedies; he should be observant of all the circumstances which render a patient susceptible, always making allowance for idiosyncrasies; and he should remember that curative effects are often obtained in the interval between physiological and toxic effects. (Seguin.) The table of doses given on page 231 indicates average doses, such as would be accepted by most authorities; but in the paragraphs which follow, some consideration will be given to the manner of using particular drugs.

The Abuse of Drugs, and their Untoward Effects.—Too little attention is usually paid to the effects on digestion of powerful hypnotics, narcotics, and sedatives. Good effects may be counteracted, in part at least, by gastric and intestinal disturbances. Drugs differ much in this respect; chloralamid, for instance, has been shown to delay and paraldehyde to accelerate digestion of fibrin when given in large quantity. Putrefaction is prevented or delayed by paraldehyde, but is not influenced by chloralamid. Strong solutions of sulphonal delay digestion and weak solutions have but little effect on it, while neither retard putrefaction. (Gordon.) Hypnotics, narcotics, and sedatives are much abused, particularly in the treatment of neurasthenia, hysteria, mania, and melancholia. Chapin has called attention to the evidences of the abuse of drugs presented by patients on their admission to hospitals for the insane, in dilated sluggish pupils, diminished mental reflexes, feeble heart beat, flabby pasty tongue, and tumid stomach. The routine hospital methods of administering such drugs are also well known, but are happily going out of use. The accumulation of drugs in the system must be carefully considered. Potassium bromide, for example, is eliminated very slowly, and bromism is not infrequently a serious complication in the treatment of epilepsy. Preparations of opium are not usually well borne by children; but bromides can be given in larger proportionate doses than to adults. Idiosyncrasies should not be disregarded. As drugs used in nervous diseases include the most powerful remedies of the pharmacopeia, not only their physiological but also their special untoward effects should be thoroughly understood. Kiernan and Baum have presented in tabular form excellent summaries of these untoward effects, from which the table (page 233) of some of the important and newer remedies has been condensed.

Untoward Effects of some of the More Potent Drugs.

Drugs.	General, Lungs, Heart.	Brain and Cord.	Eye, Ear, and Throat.	Skin, Liver, Kidneys, Bladder.
Acetanilid or antifebrin.	Cardiac failure, edema of lungs, cyanosis, fever, dyspnea, pallor, hyperidrosis, nausea, diarrhea.	Anesthesia, hyperesthesia, vertigo, headache, delirium, formication, stupor, ataxic symptoms.	Amblyopia, throat irritation, edema of glottis, tinnitus, dilated pupils, conjunctivitis.	Erythema, papules, pustules, vesicles, icterus, albuminuria, cystitis.
Amyl nitrite.	Cyanosis, collapse, pallor, hyperidrosis, nausea.	Vertigo, stupor, hallucinations, spasmodic cough, hyperesthesia, anesthesia.	Yellow vision, amblyopia, throat irritation.	Vesicles, erythema, cystitis, cystalgia.
Antipyrin.	Nausea, collapse, syncope, hyperidrosis, fever, cardiac failure, dyspnea, diarrhea.	Anesthesia, hyperesthesia, convulsive phenomena, ataxic symptoms.	Conjunctivitis, amblyopia, throat edema, tinnitus.	Erythema, vesicles, papules, pustules, green urine, cystitis, albuminuria, glycosuria, icterus.
Caffeine.	Cheyne-Stokes breathing.	Delirium, hallucination.	Diplopia.	Erythema, dysuria.
Cannabis Indica.	Aphrodisia, vomiting, diarrhea, amenorrhea.	Vertigo, delirium, ataxic symptoms.	Amblyopia, tinnitus.	Erythema, dysuria.
Chloral hydrate, chloralamid, and chloralose.	Dyspnea, bronchitis, pneumonia, heart failure, purpura, fever, hyperidrosis, diarrhea.	Vertigo, ataxic symptoms, stupor, delirium, convulsive attacks, anesthesia, analgesia, hyperesthesia.	Conjunctivitis, edema of epiglottis, amblyopia, yellow vision, photophobia, choroiditis, tinnitus.	Erythema, acne, pustules, papules, purpura, baldness, urticaria, pruritus, icterus, albuminuria, glycosuria, strangury.
Cocaine.	Anaphrodisia, collapse, diarrhea, constipation, nausea, fever, amenorrhea, tenesmus.	Vertigo, delirium, ataxic symptoms, hyperesthesia, transitory frenzy.	Amblyopia, diplopia, throat irritation, conjunctivitis.	Erythema, papules, urticaria, vesicles, icterus, glycosuria.
Conium.	Vomiting, nausea.	Vertigo, ataxic symptoms, hyperesthesia.	Amblyopia.	Erythema, dysuria.
Duboisine.	Collapse, vomiting, diarrhea, fever.	Vertigo, delirium, local hyperesthesias.	Conjunctivitis, pupil immobility, throat irritation.	Erythema, icterus.
Exalgin.	Cyanosis, fever, hyperidrosis, collapse, dyspnea.	Coma, delirium, ataxic symptoms, anesthesia, vertigo.	Amblyopia.	Erythema, icterus.
Gelsemium.	Dyspnea, coryza.	Vertigo.	Amblyopia.	Erythema, strangury, icterus.
Glonoin.	Coryza.	Vertigo, ataxic symptoms, analgesia.	Amblyopia.	Erythema, strangury, glycosuria.
Hydrastin.	Collapse, syncope, fever, hyperidrosis, heart failure.	Vertigo, ataxic symptoms.	Amblyopia, conjunctivitis.	Erythema, purpura, strangury, green urine.
Hyoscyamus.	Dyspnea, cardiac irritability, fever, nausea.	Vertigo, delirium, ataxic symptoms, numbness.	Pupil immobility, amblyopia, throat irritability.	Erythema, pustules, icterus, bladder irritability.
Methylal.	Hyperidrosis, collapse.	Vertigo, stupor, ataxic symptoms.	Diplopia.	Erythema, urticaria, bladder irritability.
Methacetin.	Hyperidrosis.	Vertigo.	Amblyopia.	Erythema. bladder irritability.
Paraldehyde.	Dyspnea, gastralgia, diarrhea, cyanosis, nausea.	Vertigo, stupor, delirium, paresthesia, ataxic symptoms.	Amblyopia.	Erythema, papules, nail ulceration, purpura, glycosuria, icterus.
Phenacetin and phenocoll.	Fever, collapse, hyperidrosis.	Vertigo, hyperesthesia.	Dim vision, diplopia, tinnitus.	Erythema, papules, dysuria, icterus.
Physostigma.	Nausea, vomiting, gastralgia.	Vertigo, ataxic symptoms.	Tinnitus, dim vision.	Urticaria, erythema, dysuria.
Sulphonal, tetronal, and trional.	Collapse, dyspnea, hyperidrosis, cardiac irritability, nausea, diarrhea.	Vertigo, stupor, paresthesia, ataxic gait, formication.	Amblyopia, tinnitus.	Erythema, papules, pruritus, urticaria, bladder irritability.

Hypnotics.—*Amylene hydrate,* one of the new hypnotics, is somewhat similar in its action to chloral and paraldehyde. It quiets cerebral excitement, and also the excitability of the spinal cord and oblongata, and has some effect on the sensory nerves. It can be given in doses of half a drachm to a drachm, or even more, under special circumstances, and in delirium tremens and in the insomnia of some forms of insanity it is of great value. *Chloralamid* is a hypnotic of proved value, safe in proper doses, and rarely depressing. In ordinary cases it stimulates the respiratory centres, the blood pressure remaining uninfluenced. It is especially useful in insomnia accompanying the high arterial tension of Bright's disease. When given for hypnotic effect its physiological action is noticed in from thirty to ninety minutes, and the sleep induced lasts from five to nine hours, is natural and refreshing, and is not followed by any unpleasant sequelæ. *Duboisine sulphate* is both hypnotic and sedative, belonging to the same order of drugs as hyoscine hydrobromate and atropine, and is useful in some forms of mental disease with excitement, as in mania, agitated melancholia, agitated dementia, and choreic insanity. It can also be used in short convulsive hysterical attacks. The association of a sedative with a somnifacient effect sometimes makes it particularly useful. It has been given hypodermatically in doses of one one-hundredth to one sixtieth of a grain, but the former is probably the safer dose for this method of administration. *Hypnal,* a hypnotic whose action is much like that of chloral and antipyrin, is known in two combinations, one *monochloral of antipyrin,* which has forty-seven parts of chloral to fifty-three of antipyrin ; the other *bichloral of antipyrin,* which has sixty-six parts of chloral and thirty-four of antipyrin. Injected into the veins of a dog, the two combinations had the same toxic "coefficient,"—one gramme for every kilogramme of the weight of the animal. (Gley.) It has hardly any taste, and no odor, which makes it superior to chloral, particularly for children. It produces hypnotic effects in smaller doses than those of chloral. The properties of its component parts (chloral and antipyrin) are manifest in its action ; thus it is both hypnotic and analgesic. It is serviceable in insomnia due to pain. (Fränkel.) *Hyoscine hydrobromate* has secured a prominent place in the armamentarium of the neurologist and alienist. It is especially useful in insomnia with delirious excitement. *Methylal* is a hypnotic of some virtue, but on the whole inferior to chloral, hypnal, or somnal. *Paraldehyde* is a nearly pure hypnotic, and one that is undoubtedly effective, and less dangerous to the heart than chloral. Its nauseous taste and the gastric disturbances produced by it are objections to its use, but these can be partly overcome by giving close attention to the form of prescription. It may be administered in drachm doses. *Somnal,* like sulphonal and trional, is a hypnotic that has probably come to stay. It will induce sleep when loss of sleep is not

dependent upon pain. In the sleeplessness of mental strain or anxiety, of hysteria or mild mania, and that which occurs after acute diseases, it is especially indicated; but it is of little value in the insomnia of organic disease of the brain. Although *sulphonal* occasionally produces untoward effects, it is an invaluable hypnotic, but the method of using it must be carefully considered. As is well known, its effects often come on late, and therefore it is best to administer it some hours before the time when it is needed to produce sleep, as in several doses of from five to ten grains, beginning in the middle of the afternoon and repeating every two or three hours. The plan suggested by Mairet, of administering large doses for a day or two and then smaller doses, is sometimes a good one; thirty to forty grains or even more may be given for one or two evenings, and then doses of fifteen grains or less every evening can be continued. Another drawback, besides its slowness in promoting sleep, is the tendency of its hypnotic action to continue the next day. Occasionally a patient shows a striking idiosyncrasy against its use. *Trional* is a safe and valuable hypnotic when given in appropriate doses. It can be administered in water, wine, or milk, and acts promptly, differing in this respect from sulphonal. *Tetronal* is a drug allied to trional and probably of nearly equal value. Among remedies which have attained some reputation as hypnotics, but have not been proved to be of sufficient value to rank with drugs like chloral, sulphonal, trional, chloralamid, and even hypnal and somnal, may be mentioned *chloralose*, *urethan*, and *ural* or *uralium*.

Analgesics.—Analgesics act differently on different parts of the nervous system. General anesthetics and local anesthetics alleviate pain, the latter by preventing the reception of painful stimuli, and the former by acting upon nerve centres so as to abolish the perception of pain. Pain can also be prevented by hindering the transmission of sensory impressions in the spinal cord. Cocaine acts energetically upon nerve trunks when locally applied or when injected subcutaneously, and affects both the conductivity and the reflex irritability in the spinal cord. Atropine, veratrine, and other local analgesics have a distinct local anesthetic action. Acetanilid and antipyrin act powerfully upon the spinal cord. (Brunton.) Duquesnel's *aconitia* or *aconitine*, if used properly, is often efficient in the treatment of neuralgic affections, and especially of trigeminal neuralgia. Patients should first be tested with small doses, as one four-hundredth to one two-hundredth of a grain, and then the dose can be pushed up to one one-hundredth two or three times daily. I have had both successes and failures with this drug. Seguin has strongly recommended its use in trigeminal neuralgia. With this remedy he recommends red iodide of mercury to be given, the dose gradually increased from one twentieth to one fifth or one sixth of

a grain, combined with potassium iodide. Aconitine is a remedy of considerable value in exophthalmic goitre, exerting a powerful influence on nervous fast pulse. Success has been achieved in the use of *agathin* in the treatment of sciatic and supraorbital and some other forms of neuralgia, but it often fails. *Codeine sulphate* is of value in the treatment of the opium or morphine habit. After first reducing morphine to the minimum, codeine can be substituted, at first giving doses of about one grain and gradually reducing. This is continued from ten to fourteen days. Acetanilid, bromo-caffeine, and quinine are used as auxiliaries. Codeine has peculiar power in lessening the irritability of the intestinal nerves, and possibly also of those of the respiratory organs. It is therefore useful in nearly all forms of abdominal pain, like colic, gastric, renal, and ovarian neuralgia. *Exalgin* has some virtues as sleep producer, pain reliever, and suppressomotor. In chorea it has been used with success. It is said to act excellently as an analgesic in children. *Methylene blue* has been used in the treatment of facial neuralgia, angiospastic migraine, nervous headache, muscular rheumatism, and herpes zoster. It is of use only in purely nervous pain. *Thymacetin* is a derivative of thymol, analogous to phenacetin: it is a white crystalline powder, slightly soluble in water. In hemicrania and in habitual headache and neuralgic pains the action is similar to that of phenacetin, rapid and certain in some cases, slight or absent in others. In headache due to organic brain lesion it is uncertain. Other drugs which have obtained some reputation for the relief of pain are *bromamide*, *phenocoll*, and *pyrodin*.

Conium.—Conium and its preparations, when reliable, have an important place in neurological therapeutics. Attention should always be paid to the source from which the drug is obtained, for, like other vegetable neurotics, it may deteriorate by keeping, and sometimes the preparations, as originally made, are inert. Physiological experiments have proved that coniine acts directly upon the efferent or motor nerves, paralyzing these nerves, although it has some but much less decided effects upon the sensory nerves. It is probably also a powerful spinal depressant. My clinical experience teaches me that it has some quieting effect upon the cerebral hemispheres. The fluid extract of conium, the preparation chiefly used by me, is of value in hysterical excitement, in common acute mania, and in chorea. In the treatment of mania it can be combined with the bromides and used alternately with hyoscine hydrobromate. In chorea I usually give the fluid extract in equal dose with Fowler's solution of arsenic, beginning with two or three minims of each, and increasing day by day until quieting or constitutional effects are produced. It makes a useful addition to a bromide mixture in the treatment of epilepsy. I have had a limited experience in the use of hydrobromate of coniine, and believe it to be a valuable drug.

The dose usually given by me is from $\frac{1}{50}$ to $\frac{1}{20}$ grain, but I have given as much as $\frac{1}{10}$ grain, and it can probably be used in still larger doses. Seguin recommends as much as a drachm of the fluid extract of conium as a dose. In the treatment of insomnia, general irritability, localized spasms, and chorea such doses cause almost immediate toxic effects, and are safe only when patients can be carefully watched. It is best to begin with four or five minims and rapidly to increase until fifteen minims or more are taken and the desired effect is produced.

Gelsemium.—Among powerful remedies of the motor depressant group is *gelsemium.* It is a drug which should be used with caution, and yet one that needs to be administered in efficient dose in order to obtain a good result. Some individuals show special idiosyncrasies against gelsemium. Sinkler has reported a case in which five drops of the fluid extract of gelsemium produced alarming toxic symptoms in a patient who had been taking the remedy three times daily for about two weeks; and in another case a dose of seven drops caused visual disturbances and unsteadiness of gait. Bassette has reported a case of chronic spasm of the muscles supplied by the spinal accessory nerve, in which the patient was first placed upon the fluid extract of gelsemium in doses of five drops four times daily, and this dose was increased daily by one drop until twenty-four drops four times daily were taken, up to which time no constitutional effects were visible. She was then placed upon Parke, Davis & Company's normal liquid gelsemium, beginning with five drops three times daily. The dose of this was increased until she took twenty drops, when she began to see double and to feel dizzy. The drug was omitted for one day, and she was then again put upon twenty drops, and this was increased to twenty-five with no effect. (Two drops of the fluid extract are equal to one minim.) For three weeks this patient took for the greater part of the time twenty-six drops, or thirteen minims, every two hours, night and day; for another part of this time, alternately, either thirteen minims or seven and a half minims, every two hours, or about this amount. For the remaining forty-seven days she took about thirteen minims eight times daily.

Antispasmodics.—Although sometimes derided, we have in the old fashioned antispasmodics remedies which are of real service in the treatment of functional nervous disorders. Some of these antispasmodics are asafetida, valerian, sumbul, cimicifuga, and musk; the last is too expensive for ordinary use, but where expense is no object it may be used with decided effect in hiccough, hysterical attacks, and conditions of extreme nervous exhaustion. Sumbul has a value fairly comparable to that of asafetida and valerian, although some specimens of the drug are valueless. It may be used in the form of a tincture, fluid extract, or solid extract. When

administered in the fluid form it is best given alone, diluted with water at the time of its administration; or the fluid extract may be combined with simple elixir. The pill long known as Goodell's pill, or the compound sumbul pill, is a convenient method of administering this drug; or it may be given in combination with salts of valerian, or preparations of asafetida or hyoscyamus, without arsenic or iron.

Cardiants.—Not infrequently, patients suffering from nervous disease, functional or organic, have a weak or diseased heart and slow and sluggish circulation. The remedies most useful in such conditions are preparations of *caffeine*, *cactus*, *digitalis*, and *strophanthus*. It is not necessary for me to dwell upon the virtues of digitalis and strophanthus. *Cactus grandiflorus* is less used and its virtues are less well known, but it is an admirable stimulant to the vasomotor centres. It is valuable in some forms of nervous cardiac palpitation. I usually give the fluid extract in doses increasing from five to ten minims. *Caffeine* has also a stimulating effect on the heart muscle and nerve centres, increasing blood pressure. It is invaluable in some forms of nervous headache, in various depressive states of the nervous system, in the cardiac neuroses, and in heart disease associated with kidney affections.

Alterative Tonics.—When in neurological practice an alterative tonic effect is desired, a good preparation is a combination of the syrup of hydriodic acid and compound syrup of the hypophosphites, in the proportion of equal parts, or of one part of the former to two parts of the latter. Small doses of sodium iodide can be added. Such a combination will be found of value in cases of neurasthenia and melancholia with hepatic and intestinal derangements, in old syphilitics, and in neuritis which has become chronic. Turpentine has been too much discarded in neural affections, such as chronic sciatica and lumbar or lumbosacral neuritis or myositis, commonly called lumbago, in which it may be the most efficient remedy that can be administered. It should be given in doses of fifteen minims or half a drachm, or even more, in a properly prepared emulsion, and a careful watch should be kept for such untoward effects as strangury. Arsenic and mercury are sometimes valuable in similar cases, and may be given advantageously in the old fashioned Donovan's solution, or in one of the two, three, or four chloride mixtures.

Metallic Tonics.—The metallic tonics have an old and a well deserved reputation in the treatment of chronic nervous diseases, both functional and organic; and gold, silver, zinc, copper, arsenic, iron, and other metals furnish salts of great value. Sodium and gold chloride, although perhaps it has attained an unmerited reputation in some directions, is a valuable tonic, and I not infrequently use it in the treatment of hysteria and other affections, usually after the manner long since recommended by Niemeyer, giving pills of

one tenth or one twelfth of a grain three times daily, and increasing the number of pills until three are taken three times daily. Both the nitrate and the oxide of silver are remedies of great value. Silver nitrate has long borne a good reputation in the treatment of sclerosis, and formerly it was much employed for epilepsy. It acts beneficially in the degenerative nervous diseases apparently by retarding the progressive pathological process and giving tone and strength to that part of the neuraxis which is still intact. The great objection to its use is the fact that it may cause argyria, or discoloration of the skin. It is difficult, if not impossible, to know just how far to go in the administration of such a drug before producing discoloration. Probably the advice given by Wood, to suspend its administration for one week at the end of every third week, and not to extend its use over a longer time than three months without a protracted intermission, is as near a practical rule as can be obtained. Silver oxide may be used instead of the nitrate, in doses of about one grain. The salts of copper, formerly used to a considerable extent in the treatment of hysteria, have gone largely out of use; but these preparations are of undoubted value as nerve tonics. They may be used with advantage in cases of hysteria or melancholia associated with neurasthenia. These preparations stimulate the circulation as well as act as nerve tonics, and are worthy of trial in hysteria, chorea, and allied affections. Barium chloride formerly bore a favorable reputation in the treatment of scleroses and of epilepsy. I have had some experience in its use, but believe that it is not of sufficient value to recommend it in preference to any of the better known drugs employed for these affections. A number of well known therapeutists have published physiological studies of this drug which show that it is of value in cases of failure of the heart muscle. It should be prepared of the strength of five grains to the ounce of water.

Phosphorus.—Doubtless some preparations of phosphorus, and phosphorus itself, are useful remedies in the treatment of nervous diseases. As already shown, complex derivatives of phosphoric acid are present in considerable amount in nervous tissues, and especially in the brain. Seguin summarizes the facts with reference to phosphorus as a neurotherapeutic agent as follows: "Phosphorus should be given pure, in the shape of a solution in alcohol and glycerin (Thompson's solution or tinct. phosphori, ʒj–$\frac{1}{20}$ grain), or dissolved in oil (oleum phosphoratum), or as pil. phosphori. The pills in the market give altogether too small doses of phosphorus, which should be administered in doses varying from $\frac{1}{60}$ of a grain to $\frac{1}{20}$, three times a day,—the oil and pills after food, the tincture (diluted, if necessary, with glycerin) on an empty stomach and without water. Food, however, conveys an appreciable amount of phosphorus into the system in a naturally assimilable state." The oil of phosphorus of

the Prussian pharmacopeia is one of the most efficient preparations of phosphorus, and was much used at the out-door neurological service of the hospital of the University of Pennsylvania when I was connected with that department.

Bromides.—Such preparations as the bromides of arsenic, zinc, iron, magnesium, and calcium have proved of little value in my hands. Better results are to be obtained from the use of the well known bromides with Fowler's solution, or the iron, arsenic, zinc, etc., separately. In the long continued use of bromides, whether for epilepsy, migraine, or whatever the disease, it is of vital importance to study the patient as well as the disease. Susceptibility to the bromides is increased by the existence of organic, cardiac, and cerebral disease. Moderate doses in such cases will sometimes produce profound bromism. The hygiene and diet of patients taking bromides are of the greatest importance. Patients under their use, and nervous patients in general, are much benefited by the use of cod liver oil and of fatty foods. Preparations of malt are also of great value, and maltine with peptones, as recommended by Graeme M. Hammond, may be found of service in epileptic and other patients suffering from exhaustive diseases or from the effect of depressing drugs. With bromides, as with other potent remedies, the dose can sometimes be regulated by a consideration of the body weight of the patient. Thus, Cesare Agostini found that in the great majority of cases, with the average dose of ten to fourteen grammes, intermitting every third day, a quantity corresponding to twenty to twenty-five centigrammes per hectogramme of weight of the individual, harmless in physiological experiments, produced a cessation or a notable diminution of convulsive attacks. Among organic bromine compounds, *ethylene bromide* has something to be said in its favor. Donath believes that it is less harmful than the bromine salts commonly used, because it does not contain the alkalies to which the deleterious effects of the latter are probably due. As it is insoluble in water, but mixes readily with alcohol and the fixed oils, it is best administered in five per cent. mixture with some bland oily emulsion or in spirit of peppermint, well diluted. The dose is from one to three decigrams, two or three times a day. For children of eight or ten years the dose of five per cent. emulsion is from ten to twenty drops, two or three times a day. In the use of the bromides, as of the iodides, the occurrence of acne cannot be depended upon to guide the dosage. This eruption may be dependent upon some idiosyncrasy or peculiarity of the patient and an unhealthy state of the skin, with over development of the sebaceous glands, and deficient excretion in other channels than the skin. Large dilution of the bromides in alkaline water, and full doses of arsenic at intervals, will do much to control the acne. (Seguin.)

FORMULAS. *

Instead of discussing further in a general way the special uses and methods of administration of drugs most frequently used in neurological practice, I shall give a series of formulas for the internal, external, and hypodermatic administration of many of these drugs, in some instances with brief comments on their uses and methods of preparation. Not a few of these prescriptions may be found useful in several, perhaps many, nervous affections.

Amylene Hydrate.

			C.c.	
℞	Amylene hydratis,	f℥iij;	11	25;
	Extracti glycyrrhizæ fluidi,	f℥ss;	15	00;
	Aquæ destillatæ, q. s. ad	f℥vj.	180	00.

M.

Sig.—Tablespoonful, in a wineglassful of water, at bedtime, or as directed.

Amylene hydrate can also be administered hypodermatically in chloroform water; but an objection to this method of administration is the comparatively large dose required.

Sodium Bromide.

			Gm. et c.c.	
℞	Sodii bromidi,	℥jss;	45	00;
	Aquæ destillatæ,	f℥vj.	180	00.

M.

Sig.—Teaspoonful three or four times daily.

Strontium Bromide.

			Gm. et c.c.	
℞	Strontii bromidi,	ʒvj;	24	00;
	Aquæ cinnamomi,	f℥vj.	180	00.

M.

Sig.—Dessertspoonful or tablespoonful, in a wineglassful of water, three times daily.

Strontium bromide acts well in some cases of epilepsy in doses of fifteen grains to a drachm, and seems to be better borne by the stomach than some of the other bromides.

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* In the formulas both the customary and the metric weights and measures have been given. In turning one into the other, Remington's method of conversion has been adopted, to avoid inconvenient fractions. The table of equivalents given by him is as follows:

gr. j =	about	0.06 gm.	(64.7989 mg.).
gr. xv =	"	1 gm.	(97.189 cg.).
ʒj =	"	4 gm.	(3.8879 gm.).
℥j =	"	30 gm.	(31.10348 gm.).

In liquid measure one minim is considered the equivalent of 0.06 cubic centimetre, one ounce of 30 cubic centimetres, and one drachm of 3.75 cubic centimetres.

In the prescriptions which follow are many new words, the names of newly introduced drugs. It has been considered best not to give a genitive to some of these words.

Chloral with Bromide.

		Gm. et c.c.
℞ Chlorali hydratis,	f℥ss;	15 | 00;
Sodii bromidi,	℥j;	30 | 00;
Aquæ destillatæ,	f℥vj.	180 | 00.

M.

Sig.—Teaspoonful three or four times daily.

Bromides with Conium and Fowler's Solution.

		Gm. et c.c.
℞ Potassii bromidi,	ʒiij;	12 | 00;
Sodii bromidi,	ʒij;	8 | 00;
Lithii bromidi,	ʒj;	4 | 00;
Extracti conii fluidi,		
Liquoris potassii arsenitis,	āā fʒss;	1 | 88;
Elixiris aromatici,	f℥ij;	60 | 00;
Aquæ destillatæ, q. s. ad	f℥vj.	180 | 00.

M.

Sig.—Tablespoonful, in a wineglassful of water, three times daily, after meals.

Hydrobromic Acid.

		C.c.
℞ Acidi hydrobromici,		
Syrupi limonis,		
Aquæ destillatæ,	āā f℥ij.	60 | 00.

M.

Sig.—One or two teaspoonfuls, in a small tumblerful of water, three times daily, half an hour after meals.

Chloral, Morphine, and Atropine.

		Gm. et c.c.
℞ Atropinæ sulphatis,	gr. ⅕;	0 | 012;
Morphinæ sulphatis,	gr. iv;	0 | 24;
Chlorali hydratis,	ʒiij;	12 | 00;
Aquæ destillatæ,	f℥j.	30 | 00.

M.

Sig.—Twenty drops in water, as directed.

Chloralamid.

		Gm. et c.c.
℞ Chloralamid,	ʒiij;	12 | 00;
Tincturæ cardamomi compositæ,	f℥j;	30 | 00;
Elixiris aromatici, q. s. ad	f℥iij.	90 | 00.

M.

Sig.—Dessertspoonful to tablespoonful, before bedtime, or as directed.

Chloralose.

		Gm. et c.c.
℞ Chloralose,	ʒj;	4 | 00;
Alcoholis,	fʒij;	7 | 50;
Syrupi aurantii,	f℥j;	30 | 00;
Aquæ, q. s. ad	f℥iij.	90 | 00.

M.

Sig.—Dessertspoonful or tablespoonful, in a wineglassful of water, before bedtime, or as directed.

Chloralose is most frequently given in capsules.

Hyoscine Hydrobromate.

		Gm. et c.c.
℞ Hyoscinæ hydrobromatis,	gr. ¼;	0 | 015;
Aquæ destillatæ,	f℥ss.	15 | 00.

M.

Sig.—Five to ten drops as directed.

Hypnal.

		Gm. et c.c.
℞ Hypnal,	ʒiss;	6 | 00;
Syrupi aurantii,	f℥j;	30 | 00;
Aquæ destillatæ, q. s. ad	f℥iij.	90 | 00.

M.

Sig.—Tablespoonful at bedtime, or as directed.

Hypnone.

		C.c.
℞ Hypnone,	fʒss;	1 | 88;
Alcoholis,	fʒij;	7 | 50;
Syrupi aurantii,	f℥ij;	60 | 00;
Aquæ destillatæ, q. s. ad	f℥vj.	180 | 00.

M.

Sig.—Tablespoonful, in a wineglassful of water, as directed.

As hypnone slows respiration and reduces blood pressure, it should be administered with care. It has a pungent taste, and therefore it is best to give it well diluted, as in the above prescription.

Methylal.

		C.c.
℞ Methylal,	fʒvj;	22 | 50;
Syrupi rubi idæi,	f℥j;	30 | 00;
Aquæ destillatæ, q. s. ad	f℥iij.	90 | 00.

M.

Sig.—Dessertspoonful or tablespoonful, in a wineglassful of water, as directed.

Paraldehyde.

		C.c.
℞ Paraldehyde,	fʒj;	3 | 75;
Tincturæ vanillæ,	♏xij;	0 | 72;
Alcoholis,	fʒv;	18 | 75;
Aquæ destillatæ,	fʒiij;	11 | 25;
Syrupi, q. s. ad	f℥iij.	90 | 00.

M.

Sig.—One to two tablespoonfuls at bedtime.

Somnal.

		C.c.
℞ Somnal,	fʒvj;	22 | 50;
Spiritus frumenti,	f℥ss;	15 | 00;
Aquæ destillatæ, q. s. ad	f℥iij.	90 | 00.

M.

Sig.—Dessertspoonful, in a tablespoonful of water, at bedtime, or as directed.

Sulphonal.

		Gm.
℞ Sulphonal,	ʒij.	8 | 00.

Ft. chart. no. viii.

Sig.—One powder to be taken in hot water or hot milk, as directed.

Sulphonal is insoluble in water at ordinary temperature, but moderate doses will partially dissolve in hot water. When the use of a small amount of alcohol is desirable or is not objectionable, sulphonal may be given with whiskey and water, using one or two drachms of whiskey. Milk to some extent disguises its unpleasant chalky taste. It may also be suspended in mucilage of acacia and given in tablespoonful or wineglassful doses.

Tetronal.

		Gm. et c.c.	
℞ Tetronal,	ʒiij;	12	00;
Spiritus frumenti,	f℥j;	30	00;
Elixiris aromatici, q. s. ad	f℥iij.	90	00.

M.

Sig.—Dessertspoonful or tablespoonful, in a wineglassful of water, before bedtime, or as directed.

Tetronal can be given in powders, in capsules, or with water or milk.

Trional.

		Gm. et c.c.	
℞ Trional,	ʒiij;	12	00;
Spiritus vini gallici,	f℥j;	30	00;
Elixiris aromatici, q. s. ad	f℥iij.	90	00.

M.

Sig.—Dessertspoonful or tablespoonful, in a wineglassful of water, before bedtime, or as directed.

Trional, like sulphonal and tetronal, can be given in the form of powders.

Uralium.

		Gm. et c.c.	
℞ Uralii,	ʒjss;	6	00;
Alcoholis,	fʒjss;	5	63;
Syrupi rubi idæi,	f℥j;	30	00;
Aquæ destillatæ, q. s. ad	f℥iij.	90	00.

M.

Sig.—Tablespoonful at bedtime, or as directed.

Physostigma.

		Gm.	
℞ Extracti physostigmatis,	gr. v;	0	30;
Extracti gentianæ,	ʒss.	2	00.

M. et div. in pil. no. xxx.

Sig.—One pill three or four times daily.

Agathin.

		Gm. et c.c.	
℞ Agathin,	ʒij;	8	00;
Spiritus vini gallici,	f℥ss;	15	00;
Elixiris aromatici, q. s. ad	f℥iij.	90	00.

M.

Sig.—Dessertspoonful in a wineglassful of water, every three or four hours.

Bromamide.

		Gm.	
℞ Bromamide,	ʒij.	8	00.

Ft. capsul. no. xij.

Sig.—One every three hours.

Bromamide has some anodyne as well as antipyretic properties.

CODEINE SULPHATE.

			Gm. et c.c.	
℞	Codeinæ sulphatis,	gr. xij;	0	72;
	Elixiris aromatici,	f℥j;	30	00;
	Aquæ destillatæ, q. s. ad	f℥iij.	90	00.
M.				

Sig.—Teaspoonful every four hours.

In the treatment of the morphine habit, either pure codeine, or the sulphate, phosphate, or muriate, is used. The pure codeine is not suitable for hypodermatic administration. The phosphate is the most soluble. (Mattison.) The dose given in the above prescription is one-half grain; but a much larger initial dose can be given in the morphine treatment.

EXALGIN.

			Gm. et c.c.	
℞	Exalgin,	ʒss;	2	00;
	Spiritus vini gallici,	f℥j;	30	00;
	Aquæ destillatæ, q. s. ad	f℥iij.	90	00.
M.				

Sig.—Tablespoonful twice daily.

Exalgin, like other coal tar preparations, is given with brandy, whiskey, or rum, to make a more perfect solution. If this use of alcohol is objectionable, exalgin may be prescribed with aromatic elixir, which contains alcohol in a less objectionable form.

METHYLENE BLUE.

			Gm. et c.c.	
℞	Methylene blue,	ʒss;	2	00;
	Elixiris aromatici,			
	Aquæ destillatæ,	āā f℥jss.	45	00.
M.				

Sig.—Tablespoonful, in a wineglassful of water, every four hours.

Methylene blue, being soluble in about fifty parts of water, can be given in a prescription like the above; usually, however, it is dispensed in capsules.

PHENOCOLL HYDROCHLORIDE.

			Gm. et c.c.	
℞	Phenocoll hydrochloridi,	ʒij;	8	00;
	Aquæ destillatæ,	f℥iij.	90	00.
M.				

Sig.—Dessertspoonful, in a wineglassful of water, every four hours, or as directed.

THYMACETIN.

			Gm. et c.c.	
℞	Thymacetin,	ʒj;	4	00;
	Elixiris aromatici,	f℥j;	30	00;
	Aquæ destillatæ, q. s. ad	f℥iij.	90	00.
M.				

Sig.—Dessertspoonful or tablespoonful every three hours, or as directed.

Thymacetin, which acts like phenacetin, may be given in doses of about the same strength as the latter.

HYDRASTIS.

		C.c.
℞ Extracti hydrastis fluidi,	f℥j;	30 | 00;
Aquæ destillatæ, q. s. ad	f℥iij.	90 | 00.

M.

Sig.—Teaspoonful, in a wineglassful of water, every four hours, or as directed.

Hydrastinine, or some of its salts, is sometimes used hypodermatically. The sulphate is soluble in water, and can be prepared so that five minims shall equal one grain. Hydrastis and its derivatives are chiefly tonic alteratives and antiperiodics, but, having some action on the vasomotor centres and the heart, may sometimes be used in apoplexies and hyperemic states of the nervous system. When applied to the skin, or used hypodermatically, the alkaloid, *hydrastine*, is a local anesthetic: it may therefore be resorted to in local and superficial neural affections, like neuralgia or neuritis.

CANNABIS INDICA, NUX VOMICA, ERGOTIN.

		Gm.
℞ Extracti cannabis indicæ, ...	gr. vj;	0 | 36;
Extracti nucis vomicæ, ...	gr. vijss;	0 | 45;
Ergotini, ...	ʒss.	2 | 00.

M. et div. in pil. no. xxx.

Sig.—One three times daily after meals.

Cannabis indica rivals opium and cocaine in the fascinating influence it exerts over many neurotics, and therefore a cannabis habit must be carefully guarded against. It is an excellent adjuvant of bromides when both a sedative and an analgesic effect are desired. The best preparations are the solid and fluid extracts.

CROTON CHLORAL. (BUTYL CHLORAL.)

		Gm. et c.c.
℞ Croton-chlorali hydratis, ...	gr. xl;	2 | 40;
Syrupi, ...	f℥j;	30 | 00;
Aquæ destillatæ, q. s. ad	f℥iij.	90 | 00.

M.

Sig.—Two teaspoonfuls every four hours.

SALOPHEN AND PHENACETIN.

		Gm.
℞ Salophen,		
Phenacetin, ...	āā ʒj.	4 | 00.

M. et div. in chart. no. xij.

Sig.—One powder every four hours.

This would serve admirably in the treatment of acute multiple neuritis, with or without joint and cardiac complications. Sodium salicylate, cinchonidine salicylate, salol, salophen, salicylamid, and salipyrin are all remedies of determined value. Sodium salicylate, salol, and salophen are of most value in the acute and subacute stages of neuritis. Salophen seems to be securing for itself a

permanent place, because of its less poisonous nature than salol, at the same time that it contains more salicylic acid. Cinchonidine salicylate, either used alone or combined with phenacetin and small doses of strychnine, is a very efficient preparation for the chronic forms of mild or moderate localized neuritis, for which both the neurologist and the general practitioner are so often consulted.

Salipyrin.

		Gm. et c.c.	
℞ Salipyrin,	ʒiij;	12	00;
Glycerini,	f℥ss;	15	00;
Syrupi rubi idæi,	f℥j;	30	00;
Aquæ destillatæ, q. s. ad	f℥iij.	90	00.

M.

Sig.—Dessertspoonful every two, three, or four hours.

Salipyrin can be given also in capsules.

Sodium Salicylate, Bromide, and Iodide.

		Gm. et c.c.	
℞ Sodii salicylatis,			
Sodii bromidi,			
Sodii iodidi,	āā ʒij;	8	00;
Aquæ destillatæ, q. s. ad	f℥vj.	180	00.

M.

Sig.—Dessertspoonful, in a wineglassful of water, three times daily.

Oil of Gaultheria.

		C.c.	
℞ Olei gaultheriæ,	fʒij;	7	50;
Mucilaginis acaciæ,			
Syrupi,			
Aquæ destillatæ,	āā f℥ij.	60	00.

M.

Sig.—Dessertspoonful to a tablespoonful, in a wineglassful of water, three times daily, after meals.

The oil of wintergreen seems in some cases to act more efficiently than any of the salicylic preparations, particularly in acute, severe cases of neuritis.

Strychnine, Phenacetin, and Cinchonidine Salicylate.

		Gm.	
℞ Strychninæ sulphatis,	gr. ss;	0	03;
Phenacetin,	gr. xx;	1	20;
Cinchonidinæ salicylatis,	gr. xl.	2	40.

M. et div. in pil. no. xx.

Sig.—One pill four times daily.

Strychnine, Salol, and Cinchonidine Salicylate.

		Gm.	
℞ Strychninæ sulphatis,	gr. ss;	0	03;
Salol,			
Cinchonidinæ salicylatis,	āā gr. xx.	1	20.

M. et div. in pil. no. xx.

Sig.—One pill three or four times daily.

Zinc Bromide, Valerianate, and Oxide.

		Gm.
℞ Zinci bromidi,		
Zinci valerianatis,		
Zinci oxidi, ...	āā ʒss.	2 | 00.
Confectionis rosæ, q. s.		

M. et div. in pil. no. xl.

Sig.—One pill three times daily. (Régis.)

Sumbul and Valerian.

		C.c.
℞ Tincturæ sumbul,		
Tincturæ valerianæ, ...	āā f℥j.	30 | 00.

M.

Sig.—Teaspoonful every two hours in sweetened water.

Valerianates of Quinine, Zinc, and Iron.

		Gm.
℞ Quininæ valerianatis,		
Zinci valerianatis,		
Ferri valerianatis, ...	āā ʒss.	2 | 00.

M. et div. in pil. no. xxx.

Sig.—One to two pills three times daily.

Three Valerianates with Aloes.

		Gm.
℞ Extracti aloes aquosi, ...	gr. xv;	1 | 00;
Zinci valerianatis,		
Quininæ valerianatis,		
Ferri valerianatis, ...	āā ʒss.	2 | 00.

M. et div. in pil. no. xxx.

Sig.—One pill three times daily after meals. (Whitla.)

Compound Sumbul Pill.

		Gm.
℞ Acidi arsenosi, ...	gr. j;	0 | 06;
Ferri sulphatis exsiccati,		
Extracti sumbul, ...	āā gr. xl;	2 | 40;
Asafœtidæ, ...	gr. lxxx.	4 | 80.

M. et div. in pil. no. xl.

Sig.—One pill three times daily after meals. (Goodell.)

Sumbul.

		C.c.
℞ Extracti sumbul fluidi, ...	fʒvj;	22 | 50;
Elixiris aromatici, q. s. ad	f℥iij.	90 | 00.

M.

Sig.—One or two teaspoonfuls every four hours, as directed.

Cactus Grandiflorus and Nux Vomica.

		C.c.
℞ Extracti cacti grandiflori fluidi,		
Tincturæ nucis vomicæ, ...	āā fʒij;	7 | 50;
Aquæ destillatæ, q. s. ad	f℥iij.	9 | 00.

M.

Sig.—Teaspoonful or dessertspoonful, in a wineglassful of water, every four hours.

Sparteine.

		Gm.
℞ Sparteinæ sulphatis, ...	gr. v.	0 | 30.
Pulveris glycyrrhizæ, q. s.		

M. et div. in pil. no. xl.

Sig.—One pill every three or four hours.

This dose can be increased, when it is desired to produce a decided effect, by giving two or even three pills at one time. Sparteine is a useful drug as an adjuvant to digitalis, or sometimes used alone in neurotic cases with cardiac disease. Dr. W. E. Hughes, who has reported favorably on the use of this drug in cardiac and renal cases, believes that the dose should not be less than one quarter of a grain, three times daily, and that it may be administered hypodermatically in doses as large as one grain.

Hypophosphites with Hydriodic Acid.

		C.c.
℞ Syrupi hypophosphitum,		
Syrupi acidi hydriodici,	āā f℥iij;	90 | 00.
M.		

Sig.—Dessertspoonful, in a wineglassful of water, three times daily after meals.

Turpentine.

		C.c.
℞ Olei terebinthinæ,	f℥vj;	22 | 50;
Pulveris acaciæ,		
Pulveris sacchari,	āā ℨv;	20 | 00;
Aquæ menthæ viridis, q. s. ad	f℥vj.	180 | 00.
M.		

Sig.—Dessertspoonful or tablespoonful, in a wineglassful of water, three times daily.

Compound Arsenical Pill.

		Gm.
℞ Acidi arsenosi,	gr. j;	0 | 06;
Cinchoninæ sulphatis,	ℨj;	4 | 00;
Ferri et potassii tartratis,	gr. lxxx.	4 | 80.
M. et div. in pil. no. xl.		

Sig.—One three times daily after meals.

Gold and Sodium Chloride.

		Gm.
℞ Auri et sodii chloridi,	gr. v;	0 | 30;
Tragacanthæ,	ℨj.	4 | 00.
Sacchari, q. s.		
M. et div. in pil. no. xl.		

Sig.—One three times daily, to be increased until three are taken three times daily.

This method of using sodium and gold chloride, recommended by Niemeyer, has long been used by me in the treatment of hysteria.

Oil of Phosphorus Mixture.

		C.c.
℞ Olei phosphorati,	♏ xlviij;	2 | 88;
Olei gaultheriæ,	♏ xxiv;	1 | 44;
Mucilaginis acaciæ, q. s. ad	f℥iij.	90 | 00.
M.		

Sig.—One or two teaspoonfuls, in a wineglassful of water, three times daily after meals.

Silver Nitrate.

		Gm.
℞ Argenti nitratis,	gr. v;	0 | 30.
Extracti glycyrrhizæ,		
Pulveris glycyrrhizæ,	āā q. s.	
M. et div. in pil. no. xxx.		

Sig.—One three times daily.

PIPERAZIN.

			Gm. et c.c.
℞	Piperazin,	ʒiij;	11 | 25;
	Syrupi aurantii,	f℥ss;	15 | 00;
	Aquæ destillatæ, q. s. ad	f℥iij.	90 | 00.

M.

Sig.—Dessertspoonful to be added to two quarts of water and drunk during one day.

Piperazin ranks among the most active of uric acid solvents, and is one of the most valuable remedies, carefully used, in the treatment of gouty and lithemic patients with nervous symptoms. It is best administered alone.

ACONITE, COLCHICUM, AND BELLADONNA.

			C.c.
℞	Tincturæ aconiti radicis,		
	Tincturæ colchici seminis,		
	Tincturæ belladonnæ,	āā ♏ lxxx.	4 | 80.

M.

Sig.—Six drops every six hours until relieved.

The above will be found efficient for the relief of sciatic or other forms of neuritis dependent upon a gouty condition of the system.

LITHIUM BIBORATE AND SODIUM BICARBONATE.

			Gm. et c.c.
℞	Lithii biboratis,	gr. xx;	1 | 20;
	Sodii bicarbonatis,	ʒj;	4 | 00;
	Syrupi aurantii,	f℥j;	30 | 00;
	Aquæ destillatæ, q. s. ad	f℥iij.	90 | 00.

M.

Sig.—One or two tablespoonfuls to be taken at a time.

This is useful in acute attacks of lithiasis with nervous manifestations. The dose given can be doubled and repeated once or oftener.

GUAIAC, CIMICIFUGA, AND ERYTHROXYLON.

			C.c.
℞	Tincturæ guaiaci ammoniatæ,		
	Extracti cimicifugæ fluidi,		
	Extracti erythroxyli fluidi,	āā f℥j.	30 | 00.

M.

Sig.—Teaspoonful, in a wineglassful of water, three times daily.

When constipation coexists, an equal proportion of fluid extract of cascara is added. This prescription will be found valuable in neuralgia and various forms of neuritis. (Eshner.)

Cimicifuga, a favorite remedy for chorea with Dr. George B. Wood, is not infrequently used by me in the treatment of this affection, and of neurasthenia, hysteria, and general nervousness. The most efficient preparation is the fluid extract, which can be given in doses of from fifteen minims to one drachm. *Coca*, particularly in the various forms of the wines of coca, has had a great vogue, largely through exploitation by ambitious pharmaceutical firms. A good wine of coca has some value in temporarily stimulating and toning an exhausted nervous system, but it is a prepara-

tion likely to be abused. Next to cocaine or its salts, the fluid extract of erythroxylon coca in doses of from half a drachm to one drachm is the most efficient preparation, and can occasionally be used with advantage as a nerve tonic.

NUX VOMICA, CLOVES, CHLOROFORM, AND CARDAMOM.

		C.c.	
℞ Tincturæ nucis vomicæ, .	f℥ij;	7	50;
Olei caryophylli, ...	f℥ss;	1	88;
Spiritus chloroformi, ...	f℥ij;	7	50;
Tincturæ cardamomi compositæ, q. s. ad . . .	f℥iij.	90	00.

M.

Sig.—One or two teaspoonfuls, in a wineglassful of water, after meals. (Griffith.)

This is a useful formula in nervous dyspepsia, or in the flatulent dyspepsia which is often present in some types of neurasthenia.

COMPOUND KOLA PILL.

		Gm.	
℞ Ferri arsenatis, ...	gr. j⅓;	0	08;
Extracti nucis vomicæ,	gr. iij;	0	18;
Extracti rhei, . .	gr. xv;	1	00;
Extracti kolæ,			
Extracti cinchonæ, ...	āā ʒss;	2	00;
Pulveris kolæ, ...	ʒj.	4	00.

M. et div. in pil. no. xl.

Sig.—One pill four times daily. (Régis.)

Kola is a cardiant and cerebral stimulant, and is now sold in the forms of fluid extract, tincture, and specially prepared wines and cordials. It seems to have some of the virtues of drugs like caffeine and coca; but it has not yet obtained a settled position.

CINCHONINE SULPHATE WITH IRON.

		Gm.	
℞ Strychninæ sulphatis, ...	gr. j;	0	06;
Ferri reducti, ...	ʒss;	2	00;
Cinchoninæ sulphatis,	ʒj.	4	00.

M. et div. in pil. no. xxx.

Sig.—One three times daily.

OINTMENT OF ACONITINE AND VERATRINE.

		Gm et c.c.	
℞ Aconitinæ, ...	gr. iv;	0	24;
Veratrinæ, ...	gr. xv;	1	00;
Glycerini, . . .	f℥ij;	7	50;
Cerati, ...	ʒvj.	24	00.

M. et ft. ung.

Sig.—Apply once or twice daily.

Care should be taken to see that there is no abrasion of the skin at the place of application.

OINTMENT OF VERATRINE AND MORPHINE HYDROCHLORATE.

		Gm.	
℞ Veratrinæ,			
Morphinæ hydrochloratis, ...	āā gr. iv;	0	24;
Unguenti aquæ rosæ, ...	℥ss.	15	00.

M. et ft. ung.

Sig.—A piece the size of a pea to be rubbed over the painful nerve. (Bamberger.)

If an accurate dose is desired, the ointment can be divided into equal parts, and each part can be wrapped in paraffin paper, after the manner already described in discussing mercurial inunction.

IODOFORM OINTMENT.

		Gm.
℞ Iodoformi,	℥ss;	15.00;
Petrolati mollis,	℥ijss.	75.00.
M. et ft. ung.		

Sig.—Use as directed, once or twice daily.

COLLODION WITH CANTHARIDES.

		C.c.
℞ Collodii cum cantharide,	f℥ss.	15.00.

Sig.—Apply with camel's-hair brush, as directed.

This and the next prescription may be used as an absorbent counterirritant in localized neuritis.

IODOFORM COLLODION.

		Gm. et c.c.
℞ Iodoformi,	gr. xx;	1.20;
Collodii flexilis,	f℥j.	30.00.
M. et ft. solutio.		

Sig.—Brush upon the part as directed.

COMPOUND CHLOROFORM LINIMENT.

		C.c.
℞ Chloroformi,		
Ætheris,		
Spiritus camphoræ,		
Tincturæ opii,	āā f℥j;	30.00;
Tincturæ capsici,	f℥ss.	15.00.
M.		

Sig.—For external use as directed.

COMPOUND ORIGANUM LINIMENT.

		C.c.
℞ Tincturæ aconiti radicis,		
Olei origani,		
Spiritus camphoræ,		
Spiritus chloroformi,	āā f℥ss;	15.00;
Aquæ ammoniæ,		
Alcoholis,	āā f℥j.	30.00.
M. et ft. lin.		

Sig.—For external use.

This is a powerful counterirritating liniment which can be applied by rubbing with the hand or with flannel, and is useful in the treatment of lumbago and other neural and muscular affections.

LINIMENT OF CROTON OIL.

		C.c.
℞ Olei tiglii,		
Olei olivæ,	āā f℥j.	30.00.
Ft. linimentum.		

Sig.—For external use as directed.

CORROSIVE SUBLIMATE WASH FOR LAVAGE OF THE STOMACH.

		Gm. et c.c.
℞ Hydrargyri chloridi corrosivi,	gr. j;	0.06;
Aquæ destillatæ,	Oj.	480.00.
M.		

In cases of hyperacidity, the stomach is first washed out with the above antiseptic solution, after which simple alkaline lavage without antiseptics is used. (Régis.)

CREOLIN AND SODIUM BICARBONATE FOR LAVAGE.

		Gm. et c.c.
℞ Creolin,	gr. vijss;	0 | 45;
Sodii bicarbonatis,	gr. xlv;	2 | 70;
Aquæ destillatæ,	Oj.	480 | 00.

M. et ft. emulsio.

LACTIC ACID FOR LAVAGE.

		Gm. et c.c.
℞ Acidi lactici,	ʒijss;	10 | 00;
Aquæ destillatæ,	Oj.	480 | 00.

M.

In cases of achlorohydria and of dyspepsia from fermentation, antiseptic lavage with the above prescriptions is used, to be followed by washing with acid wash. (Régis.)

HYDROCHLORIC ACID WASH FOR LAVAGE OF THE STOMACH.

		C.c.
℞ Acidi hydrochlorici,	fʒss;	1 | 88;
Aquæ destillatæ,	Oj.	480 | 00.

M.

RESORCIN FOR LAVAGE.

		Gm. et c.c.
℞ Resorcini,	ʒss;	2 | 00;
Aquæ,	Oj.	480 | 00.

M.

SALOL FOR LAVAGE.

		Gm. et c.c.
℞ Salol,	ʒss;	2 | 00;
Aquæ,	Oj.	480 | 00.

M.

Hypodermatic Medication.—Among remedies most frequently used hypodermatically are salts of morphine and atropine, hyoscine hydrobromate, theine, cocaine, ether, chloroform, salts of strychnine, and preparations of mercury. Other remedies less frequently used but efficient, administered in this way, are duboisine sulphate, chloral, antipyrin, coniine hydrobromate, apomorphine, and preparations of arsenic, curarine, sparteine, and eserine. After some general remarks on the use of a few of these remedies, a series of hypodermatic formulas, derived from various sources, or from personal experience, will be given. Many of these drugs are sold in the form of soluble, compressed hypodermatic tablets, the list of these including preparations of the following: apomorphine, atropine, ergotin, cocaine, duboisine, eserine, hyoscyamine, mercuric chloride, morphine, morphine and atropine, nitroglycerin, pilocarpine, and strychnine. Subcutaneous injections of *ether* are valuable in the treatment of sciatica and other nerve affections; one cubic centimetre (fifteen minims) can be injected best where the nerve

trunks are few, and well covered with skin and fat. Care should be taken, as local paralysis has followed its use. Bartholow strongly advocates the deep injection of *chloroform* in the treatment of neuralgias. My own experience has been confined to a few cases of sciatica, which were greatly benefited by its use. The pain caused by the injection, and the tendency to the formation of swellings and abscesses, are objections to its employment, and as high an authority as Anstie declared it to be unfit for hypodermatic use. Bartholow, following Salkowski, gives aqua chloroformi as the vehicle in some of his hypodermatic formulas. This aqua chloroformi is simply distilled water, containing the small amount of chloroform which it will take up, this, according to Salkowski, preventing the formation of microorganisms because of its germicidal powers. Chloral hydrate is seldom called for hypodermatically, but in severe forms of convulsion, and in mania with delirious excitement and extreme insomnia, it may occasionally be resorted to with advantage, either alone or in combination with morphine, or with both morphine and atropine. The usual dose for hypodermatic medication is not more than ten grains. The pain and local irritation to which it usually gives rise are contraindications to its use, and it should be resorted to only when it cannot be efficiently employed either by the mouth or by the rectum.

DUBOISINE SULPHATE.

		Gm. et c.c.
℞ Duboisinæ sulphatis,	gr. j;	0 | 06;
Aquæ chloroformi,	f℥ss.	15 | 00.

M.

Sig.—Five minims contain nearly one fiftieth of a grain. (Bartholow.)

ESERINE SULPHATE.

		Gm. et c.c.
℞ Eserinæ sulphatis,	gr. j;	0 | 06;
Aquæ destillatæ,	f℥ss.	15 | 00.

M.

Sig.—Two minims equal one one-hundred and twentieth of a grain. Dose, from two to four minims.

HYOSCYAMINE.

		Gm. et c.
℞ Hyoscyaminæ sulphatis,	gr. ss;	0 | 03;
Aquæ destillatæ,	f℥ss.	15 | 00.

M.

Sig.—Five minims equal one ninety-sixth of a grain.

COCAINE.

		Gm. et c.c.
℞ Cocainæ hydrochloratis,	gr. iv;	0 | 24;
Aquæ destillatæ,	f℥ss.	15 | 00.

M.

Sig.—Five minims equal one twelfth of a grain.

Cocaine hydrochlorate is easily soluble in water, and can be administered hypodermatically in doses of from one twelfth to one fourth of a grain, in simple aqueous solution; or dissolved in liquid

petroleum or other similar oils. (Bartholow.) The extraordinary differences in susceptibility to this drug should be borne in mind. The minimum dose will affect one person toxically, while another will stand the maximum dose or the continuous use of the drug without unpleasant effects. The tendency to the formation of the cocaine habit should not be overlooked.

Coniine Hydrobromate.

		Gm. et c.c.
℞ Coniinæ hydrobromatis,	gr. j ;	0 | 06 ;
Aquæ destillatæ,	f℥j.	30 | 00.

M.

Sig.—Five minims contain about one ninetieth of a grain. (Bartholow.)

Authorities seem to differ considerably as to the minimal and maximal doses of this drug. The dose for administration by the mouth is given by some to be as much as from one sixth to one half a grain; by others, from one thirtieth to one fifteenth of a grain. With what is known of the drug, five to ten minims of the above solution should be safe for hypodermatic administration.

Curarine.

		Gm. et c.c.
℞ Curarinæ sulphatis,	gr. j ;	0 | 06 ;
Aquæ destillatæ,	f℥j.	30 | 00.

M.

Sig.—Five minims equal one ninety-sixth of a grain.

Curara can be used instead of curarine sulphate, preparing a solution that will contain one one-hundredth of a grain of the drug to the minim. The dose of curara is from one twenty-fourth to one sixth of a grain.

Theine. (Caffeine.)

		Gm. et c.c.
℞ Theinæ,		
Sodii benzoatis,	āā ʒj ;	4 | 00 ;
Sodii chloridi,	gr. viij ;	0 | 48 ;
Aquæ destillatæ,	f℥j.	30 | 00.

M.

Sig.—Six minims equal one half grain of theine.

Theine has been recommended by Mays for the relief of painful affections, and I have found it of value, in doses of from one to two grains, in superficial neuralgia or neuritis.

Fowler's Solution.

		C.c.
℞ Liquoris potassii arsenitis,	f℥j.	30 | 00.

Sig.—Dose, from five to twenty drops. (Bartholow.)

Sodium Arsenate.

		C.c.
℞ Liquoris sodii arsenatis,	f℥j.	30 | 00.

Sig.—Dose, from ten to thirty drops. (Bartholow.)

The tincture of lavender used in the preparation of the liquor potassii arsenitis should be omitted, because of its irritating proper-

ties when used hypodermatically. (Bartholow.) Large and what would seem under ordinary circumstances dangerous doses are recommended by some authors, but the results which have been obtained by such almost heroic treatment have certainly been good; especially in severe and obstinate cases of chorea has the hypodermatic use of the solutions of sodium arsenate and potassium arsenite proved of great value.

MERCURIC CHLORIDE.

			Gm. et c.c.
℞	Hydrargyri chloridi corrosivi, . . .	gr. j;	0 | 06;
	Aquæ destillatæ, ...	f℥j.	30 | 00.

M. et filt.

Sig.—Dose, ten minims.

Mercury, used hypodermatically, is of great value in the treatment of nervous syphilis. The bichloride is simply dissolved in water, as recommended by Bartholow, Wolff, and others. Soluble compressed hypodermatic tablets are sold containing one sixtieth of a grain of mercuric chloride and one fourth of a grain of sodium chloride. The insoluble compounds of mercury, and particularly calomel, are sometimes to be preferred in syphilis of the nervous system, as in the treatment of syphilis of other tissues and organs. The most rapid results are obtained from the soluble compounds, but the tendency to relapse is greater than after the use of the insoluble preparations. The mercury can be given with advantage in some forms of *gray oil*. An objection to the use of mercury hypodermatically is the pain which is produced; but, in spite of this drawback, many patients who realize the speedy and decided benefit obtained from the use of the remedy hypodermatically will ask for its administration in this way in preference to the slower, if less painful, method by the mouth or by inunction. Between the scapulæ and various positions in the back are convenient places for its administration. The directions here given are chiefly taken from papers by Dr. L. Wolff, of Philadelphia.

CALOMEL.

			Gm.
℞	Hydrargyri chloridi mitis,		
	Petrolati liquidi, ...	āā gr. lxvij;	4 | 02;
	Lanolini, ...	ℨj.	4 | 00.

M.

Sig.—One cubic centimetre to be injected not oftener than two or three times the first week. Each cubic centimetre contains three hundred and seventy-one milligrammes of mercury.

Calomel stands at the head of the insoluble mercurial preparations.

AMYL NITRITE, NITROGLYCERIN, AND OTHER NITRITES.

			C.c.
℞	Amyli nitritis, ...	f℥ij.	8 | 00.

Sig.—Dose, from two to five minims.

Bartholow says that for subcutaneous injection the amyl nitrite itself should be used as indicated by the above. Commonly this drug is used by inhalations, in doses of from one to five minims, dropped upon the handkerchief or upon absorbent cotton. Instead of the pure drug, which is exceedingly volatile, an alcoholic solution can be prepared for inhalation. It can also be administered by the mouth. Wood recommends to give it in this way on a lump of sugar. *Nitroglycerin* is most conveniently administered, both hypodermatically and by the mouth, in the form of compressed tablets, usually of the strength of one two-hundredth to one one-hundredth of a grain. *Spirit of glonoin*, which is sometimes preferred, is a one per cent. alcoholic solution of nitroglycerin. *Potassium nitrite* and *sodium nitrite*, which may be used for nearly the same purpose as amyl nitrite and nitroglycerin, can be given by the mouth in aqueous solution, well diluted, in doses of from two to three grains of the pure drugs.

Ergotinin.

		Gm. et c.c.	
℞ Ergotinin,	gr. iij;	0	18;
Acidi lactici,	gr. vj;	0	36;
Aquæ destillatæ,	f℥j.	30	00.

M.

Sig.—Five minims equal one thirty-second of a grain.

Ergotinin can be administered by the mouth, using a formula like the above so as to give about one thirtieth of a grain in a teaspoonful. As ergot in some form is often required in neurological practice, it is important to be able readily to command its most efficient preparations. Ergotinin is one of the most effective of these. *Ergotin* is a less definite and less efficient preparation. Kloman extols the physiological and therapeutic action of *ergotole* as compared with that of ergot, saying that the effect of the drug is more certain and more acceptable to the patient, and the hypodermatic use is less irritating and less painful. Ergotole, while nearly three times as strong as the pharmacopeial fluid extract, and representing all the active ingredients of ergot, has none of its nauseating smell, taste, or irritating properties.

Strychnine Sulphate.

		Gm. et c.c.	
℞ Strychninæ sulphatis,			
Acidi borici,	āā gr. ij;	0	12;
Aquæ chloroformi,	f℥j.	30	00.

M.

Sig.—Five minims equal one forty-eighth of a grain.

Strychnine Nitrate.

		Gm. et c.c.	
℞ Strychninæ nitratis,	gr. ij;	0	12;
Acidi borici,	gr. iij;	0	18;
Aquæ chloroformi,	f℥j.	30	00.

M.

Sig.—Five minims equal one forty-eighth of a grain.

Strychnine hypodermatically is useful in peripheral paralyses of toxic, infectious, and other origin, but not infrequently irritation of the subcutaneous tissues and even abscesses result from its use. Usually the best method of preparing the drug for hypodermatic use is simply to dissolve it in water, preferably hot water, but boric acid can be used to render it less irritating. Chloroform water can be substituted for distilled water. Strychnine is a valuable remedy in the treatment of alcoholism and of drug habits in general, having decided virtues in toning the nervous system, in removing, at least for a time, morbid cravings, and in supporting the heart and the respiratory functions during the treatment. Both the sulphate and the nitrate of strychnine can be used in doses of one forty-eighth to one twenty-fourth of a grain, or in some cases even one twelfth of a grain, according to indications. In the treatment of the morphine habit nitrate of strychnine can be administered with morphine, increasing the former and decreasing the latter rapidly, and so regulating the increase and decrease as to reach the maximal dose of strychnine, when the morphine is entirely omitted; or codeine may be used in decreasing doses instead of the morphine salt.

APOMORPHINE.

			Gm. et c.c.
℞	Apomorphinæ hydrochloratis, .	gr. ij;	0 | 12;
	Aquæ destillatæ, . .	f℥j.	30 | 00.
M.			

Sig.—Fifteen minims contain one sixteenth of a grain nearly. (Bartholow.)

Apomorphine, in doses of one tenth of a grain, has been used with success in cutting short hysterical attacks, also instead of morphine in breaking up the morphine habit.

Elæomyenchysis.—Corning has recommended the intramuscular injection and congelation of oils in the treatment of chronic local spasm. He holds that by injecting melted oil into a spastic muscle in sufficient quantity to interfere materially with the blood-flow and metabolism, then solidifying by means of cold, there would result interference with nutrition and curtailment of the action of the muscle, the hardened oil acting as a species of intramuscular splint. He has reported a case of chronic spasm of the splenius muscle in which this method was successfully tried. These injections should be made while the muscle is flexed, to allow of more even distribution, and to secure splinting in this position. If necessary, ether may be administered to accomplish this where we are confronted with severe tonic spasm. The second point of importance is the distribution of the oil, which should be made readily through the affected muscle; moderate massage greatly facilitates this part of the operation. A needle of large size provided with an ample lumen is necessary for the injection, and the syringe should hold at least half an ounce and should be heated to 110° F. before filling.

CHAPTER III.

DISEASES OF THE MEMBRANES, SINUSES, AND VEINS OF THE BRAIN, AND ENCEPHALIC MALFORMATIONS AND ABERRATIONS.

Associations and Complications.—In considering special diseases of the nervous system, difficulties at once arise, and will increase and vary in character as the work proceeds. Diseases of the membranes and sinuses of the brain, for example, must be independently treated, and yet their discussion cannot always be separated from that of affections of the scalp, the skull, the organ of hearing, and the brain itself. It will be necessary to refer again, at least incidentally, to diseases of the brain envelopes and of the sinuses or great venous bloodways which are found in them. Inflammation of the dura is frequently associated with disease of the ear, or it may be the result or the concomitant of grave blood affections. Sinus thrombosis also may spring from aural disease, and, like inflammation of the dura, may be associated with inflammation of the pia, and the latter with abscess of either the cerebrum or the cerebellum. One of the varieties of dural inflammation occurs and recurs during the course of general paralysis of the insane. Inflammation of the soft membranes in several of its forms may be conjoined with inflammatory disease of the superficial layers of the brain cortex, giving rise to meningoencephalitis. Some chronic encephalic affections, as idiocy, imbecility, and the cerebral palsies of children, can be referred to meningeal hemorrhage or to inflammations of the hard or soft membranes of the brain during infancy.

Functions of the Dura.—The dura is, next to the skull, the most important protector of the brain. It is supplied with blood from three meningeal arteries, an anterior (predural), a middle (medidural), and a posterior (postdural). The middle meningeal, by far the largest and most important pathologically, is a branch of the internal maxillary. Meningeal hemorrhage is most frequently due to lesion of this vessel. Veins accompany the arteries of the dura, although others, smaller in size, are independent and open into the sinuses. The dural blood supply is full and free. The investigations of Peli as to the relative depth, on each side of the skull, of the sulcus which lodges the middle meningeal arteries are of interest. In the sane, on the left side it was larger in sixty-five per cent. and on the right in twenty-three per cent. ; in the insane, the percentages were fifty-nine and a fifth on the left and twenty-six on the right. The dura undoubtedly possesses an im-

portant nerve supply, although, strange to say, this has been questioned. Froment, Arnold, Cruveilhier, Bonamy, and Duret have given the best descriptions of these nerves. Duret has demonstrated that their irritation may produce hyperesthesia, pain, and reflex motor and vasomotor disturbances. These nerves spring from the fifth pair, and are distributed nearer to the internal than to the external surface of the membrane, which explains why some lesions of the dura are more likely than others to lead to spasm. The difference depends, in part at least, on the site and intensity of the lesion with reference to the internal and external aspects of the membrane. Subdural hemorrhage is more likely to give rise to reflex spasms than are extravasations between the membranes and the skull, unless the blood tears through the membrane. A spicule of bone, in like manner, driven into the dura, is more likely to cause reflex dural spasms than a depressed fragment, while a tumor arising in the membrane is more likely to bring about the same result than an exostosis, or a neoplasm growing from the agglutinated membranes into the brain substance.

Functions of the Pia.—The pia is a delicate membrane, composed mainly of bloodvessels and connective tissue; practically it is a vascular mesh or network. It everywhere closely invests the brain, dipping into its fissures. It differs in function from the dura, not serving to protect the brain; in fact, not a few of the most serious lesions of the encephalon originate in or around its vessels, which may be ruptured or occluded, or may constitute the lines along which tuberculous, syphilitic, or other forms of disease are located. Its function is mainly nutritive, the vessels which compose it supplying a large portion of the brain with blood. The vessels lie on the surface of the membrane, being covered only by the thin arachnoid, now generally considered as the parietal layer of the pia. They are also encased in perivascular sheaths, constituted of denser portions of the membrane. The perivascular spaces thus formed are lymph canals, which penetrate with the vessels into the brain substance and communicate with the subarachnoid spaces or cisterns. In considering lesions of the pia, therefore, the vessels and lymphatics play an important part. The nerve supply of the pia, as of the dura, has been the subject of much investigation and difference of opinion. It is well known that affections of the pia-arachnoid are frequently attended with head pain, and yet it is doubtful whether this membrane, like the dura, has sensory nerves. In recent years it has been often shown at cerebral operations that this membrane is practically insensitive. The pain of pial disease is probably, therefore, commonly dependent upon coincident involvement of the nerves of the dura, or of the sensory nerves and ganglia at the base. Nerves, however, have been traced in this membrane by Purkinje, Koelliker, and others, and are in all probability vasomotor in function. They

have been shown to originate from the gangliated system, and from the third, fifth, sixth, seventh, ninth, tenth, and eleventh pairs. They accompany the arteries in the membranes and in the brain substance. Following the most recent anatomical authorities, in a previous section I have spoken of the arachnoid as if it were a separate membrane, but, as suggested by J. Batty Tuke, it would tend to obviate error if the term arachnoid were entirely dispensed with and the visceral and parietal layers of the pia were spoken of as we speak of similar layers of the pleura. The term *arachnopia* or *pia-arachnoid* can be used as a compromise. It must be understood that no membrane exists between the arachnopia and the dura.

Pacchionian Granulations.—Pacchionian granulations need to be borne in mind in the discussion of the diseases of the meninges. Inexperienced physicians and students, on first making autopsies, may get mistaken ideas as to the meaning of the appearances presented by them. When superabundant, closely clustered, and of large size, they probably do in many cases indicate chronic pathological disturbances. These Pacchionian granulations may appear in at least four places,—on the outside of the dura, on its inner surface, on the arachnopia, and within the superior longitudinal sinus and the parasinoidal spaces or lacs sanguins. They often indent the calvarium, and in rare instances they penetrate it. They are always found in greatest abundance along the median edges of the hemicerebrums. It is generally conceded that these granulations are enlargements of the normal villi or tuft-like elevations of the parietal layer of the pia or so-called arachnoid. In the newly born the villi are present, but they do not take the shape of Pacchionian granulations until a few years after birth. When the subarachnoidal space is injected, the fluid will pass into these Pacchionian bodies, and from them into the sinuses and parasinoidal spaces. While they distend the subdural spaces related to them, they do not penetrate into the general subdural cavity. The fluid filters through the villosities into the sinuses and parasinoidal spaces. These facts throw light on the increase in number and size of these formations in meningeal disease and in all affections which may cause increase of subarachnoidal pressure. In brain tumors, in tubercular meningitis, in chronic alcoholism, and in general paralysis of the insane, these bodies may be of large size and superabundant. Their presence and character should therefore always be noted. Repeated attacks of meningeal hyperemia probably assist in their development. It is supposed by Browning that when the parasinoidal spaces are present these granulations are not so likely to cause depressions in the skull, because they have free spaces in which to grow. Some clinical symptoms have been attributed to Pacchionian granulations, but only a few can with any positiveness be regarded as due to them.

Browning mentions cases where such granulations were near the Gasserian ganglion and the motor nerves of the eye, causing ocular neuralgia and paretic symptoms. Meyer refers various neuralgias to them. Headaches have been attributed to them in some instances, but with doubtful propriety. Possibly they may cause sinus thrombosis. They sometimes produce little flat elevations of bone along the median line of the crown of the head. Browning refers to a varix of the sinus longitudinalis which he believed developed from the parasinoidal spaces, and quotes a case of Meschede's in which a patient had suffered from epilepsy for thirty years, and after death a varix the size of a bean, which had reduced the bone to paper thickness, was found.

Classification of Diseases of the Brain Membranes.—Diseases of the brain membranes include (1) affections of the dura; (2) affections of the pia or arachnopia. Dural diseases include hyperemia, several forms of inflammation called *pachymeningitis*, hemorrhage, tumor, and abscess. Diseases of the pia include hyperemia and anemia and various forms of inflammation called *leptomeningitis*.

Varieties of Pachymeningitis.—Pachymeningitis is divided into *external pachymeningitis* and *internal pachymeningitis*. Internal pachymeningitis is subdivided into *hemorrhagic*, *purulent*, and *serous*. In rare cases the entire membrane may be involved in a *general pachymeningitis*, usually dependent upon a severe traumatism, or a virulent infection, as syphilis. From an etiological standpoint, dural inflammation, like pial, may be variously subdivided. Gussenbaur has reported a case of what he regards as primary circumscribed tubercular pachymeningitis. Although the case followed injury, Gussenbaur rejected the diagnosis of traumatic meningitis, as no symptoms appeared for four weeks, and as, after operation, microscopical examination showed, in granulations removed from the dura, miliary tubercles containing giant cells. Among so-called etiological varieties are the traumatic, solar, syphilitic, rheumatic, gouty, alcoholic, and erysipelatous, and forms of aural and nasal origin. In an old case of secondary dementia following melancholia, which had developed facial erysipelas ten days before death, extensive purulent pachymeningitis was found by me associated with leptomeningitis and sinus thrombosis. Leptomeningitis is much more likely to occur with infectious diseases, but these also at times give rise to pachymeningitis.

DURAL HYPEREMIA.

Dural hyperemia or congestion seldom exists alone, but is somewhat frequently associated with either congestion or inflammation of both the inner and outer membranes and of the brain substance. In the latter case its symptomatology cannot be differentiated from that of the other parts attacked. It may be active or passive, acute

or recurrent. The passive forms dependent upon obstruction to the circulation, as by thrombi and growths, are discussed in other connections. A form of active pachymeningeal hyperemia, with abrupt onset and rapid course, occurs in children, usually ending fatally. From the very nature of the affection, the course of any form of dural congestion is likely to be brief. The symptoms are those of the early stages of an acute pachymeningitis,—headache, vertigo, nausea, delirium, insomnia, local or general spasms, sense of fulness in the head, and tinnitus. Signs of retinal irritation and pupillary changes are sometimes present. These changes are probably due to irradiation of irritation by dural branches of the trigeminus to the basal ocular nuclei and nerve tracts. Dural hyperemia, when an independent affection, is usually due to some locally acting cause, as traumatism or sunstroke. It is difficult and usually impossible to make a positive diagnosis of pachymeningeal hyperemia, but when the symptoms given above occur abruptly or in a recurrent fashion, and other affections of the brain and its membranes can be excluded with reasonable certainty, the diagnosis may be assumed. The prognosis is relatively favorable, except in the children's affection to which reference has been made. The treatment is the same as that of acute pachymeningitis.

EXTERNAL PACHYMENINGITIS.

External pachymeningitis is inflammation of the outer layers of the dura. It is rarely, if ever, a primary disease. The most common symptoms are headache, fever with delirium, vertigo, nausea or vomiting, and sometimes local or general spasms. Paralysis or paresis, due either to reflex irritation or to pressure by the purulent effusion, has been noted in rare instances. External evidences of injury or of purulent effusion in the ear or in other parts may be present. When suppuration takes place, septic symptoms and those of compression may develop, the patient suffering with fever and delirium, and, if not relieved, perhaps dying in coma. Unconsciousness may develop early in severe cases. It is probable that very few of the symptoms of external pachymeningitis are due to the affection of the outer lamina of the dura; they are rather the evidences of the direct or indirect involvement of other parts, as the soft membranes, the brain, and some of the cranial nerves. A peculiar form of external chronic meningitis is sometimes present in old age, particularly in those who have led a dissipated life or who have been the victims of an infectious disease, as syphilis or tuberculosis. The symptoms in such cases are usually not definite, but dull headache is one of the most common, and is usually associated with some mental obtuseness and deterioration due largely to senile brain degeneration. As external pachymeningitis is generally a secondary or associated affection, its course and duration must de-

pend largely upon its causes, the most important of which are traumatisms, cranial caries, purulent disease of the ear, erysipelas of the face or scalp, and possibly infectious diseases. Sunstroke and heatstroke are occasional causes, especially in hot climates. The diagnosis is made by a consideration of the causative affections and associated conditions. The prognosis is, on the whole, unfavorable, although in the traumatic and other purulent varieties the patient may sometimes be greatly relieved or even cured by surgical procedure. The treatment is usually that of the affection giving rise to the pachymeningitis. Trephining for the removal of the purulent effusion may be effectual. When external pachymeningitis is due to syphilis or any infectious disease, its treatment should be in accordance with these indications. For the pachymeningitis of old age nothing can be done but to support the failing strength of the patient and carefully regulate the functions of the various organs.

DURAL HEMATOMA (HEMORRHAGIC INTERNAL PACHYMENINGITIS).

Definition.—Hemorrhagic internal pachymeningitis or hematoma of the dura is the only form of inflammation of the internal surface of the dura which has been well described; and even yet the symptomatology of this affection has not been defined with great clearness, although many pathological observations have been put on record. This disease reveals itself as a more or less recent, flat, meningeal blood tumor, or as a series of superimposed layers of connective tissue showing evidences of former extravasation. W. Bevan Lewis strongly inclines to the view that this affection is always, at least among the insane, of hemorrhagic origin, and therefore should not be discussed as a pachymeningitis; but, as the question is still an open one, it will be best to consider it here among the meningeal inflammations, giving the views which have been held by distinguished authorities.

Clinical History.—Apoplectic seizures may occur in the progress of this disease, although their nature is not commonly recognized. Chronic hemorrhagic pachymeningitis, as an independent affection, gives such symptoms as dull headache, vertigo, usually of the mild type, tendency to somnolence, and paresis, more or less marked, of one or both sides of the body. While the affection is usually bilateral, it is generally, even in such cases, more extensive on the one side than on the other, and hence the paresis produced by compression of the brain usually predominates in the limbs of one side. The limbs are more frequently hemiparetic than the face, which is probably accounted for by the position of the lesion. Occasionally convulsions, which may be unilateral or general, occur at intervals in the progress of the disorder. Pupillary contraction, usually on one side, may be present. Nystagmus and optic neuritis are mentioned

as symptoms by some authorities, but I have never met with them in cases which have fallen under my own observation. Conjugate deviation of the eyes and spastic conditions of the limbs are sometimes observed. Fraenkel has reported a case which was evidently chronic, but which came under his observation at the time of an apoplectic seizure. When the patient attempted to turn the eyes to the left a marked nystagmic tremor developed as soon as they reached the median position, which they did not cross. Both knee jerks were exaggerated, with left sided patellar and ankle clonus. The triceps jerks and all bone and muscle reflexes were lively in both arms. Cremaster and abdominal reflexes were abolished. Attempts at straightening the flexed arms caused tetanic spasm with opisthotonos. Three days after admission (seven from the first complaint) the patient presented spasms of the entire body, with deep coma and gradual resolution of rigidity, and disappearance of the patellar and foot clonus, but with lively knee jerk. The pupils reacted until shortly before death, which occurred on the eighth day. The temperature was normal until shortly before death. Difficulty of swallowing existed in the same case. The autopsy showed an immense recent hematoma, with chronic changes in the membranes. The duration and course are important, as they often serve to fix the diagnosis. The disease is essentially chronic, often lasting through years. At intervals apoplectic and inflammatory exacerbations take place, so that the symptoms naturally arrange themselves into those of the chronic condition and those of the special attacks. The disease is said to be acute in children who are subjects of hemorrhagic rachitis. The following summary of symptoms is chiefly as given by Huguenin, to whom we owe the most elaborate discussion of this disease: (1) the characteristic course shows an acute diffused affection of the brain with severe symptoms, an acute attack, followed by fair recovery, and intervals of comparative health; (2) evidences of a sudden and increasing compression are headache, drowsiness, loss of consciousness, fever, characteristic pulse, and sometimes violent initial symptoms of irritation; (3) the symptoms during the interval are headache, diminution of intelligence, impairment of memory, drowsiness, partial paralysis, disturbances of speech, and sudden mental excitement without cause, mixed with symptoms of paralytic dementia.

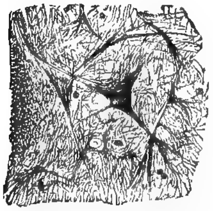
Etiology.—This disease occurs most frequently in the aged, in those prematurely senile, or in those broken down by infectious diseases or alcoholic excesses. It is of comparatively frequent occurrence in wards for chronic nervous diseases and among chronic patients in the insane hospitals. Many cases have been discovered in autopsies in the Philadelphia Hospital, most of which were not recognized during life, but had other more readily diagnosticated diseases of the viscera and bloodvessels. Chronic alcoholism, syph-

ilis, and tuberculosis are important in its etiology. It is not infrequent in general paralysis of the insane, and it is sometimes determined by the existence of diseases which affect the blood, as scurvy, small-pox, and typhus fever. It is chiefly a disease of the male sex. Cranial traumatism may be an exciting cause.

Diagnosis.—When the disease is an independent affection of the aged, the diagnosis is much assisted by a consideration of etiology and history. The occurrence of special attacks of an apoplectiform nature and the increase of the symptoms at intervals are helpful. When confronted by the problem of diagnosis at the time of a hemorrhagic attack, some of the affections which will need to be differentiated are intracranial and ventricular hemorrhage. The paralyses due to hemorrhage into the basal ganglia and capsule are usually flaccid, and such cases present peculiarities of temperature, pulse, and respiration which are significant. The longer duration of the symptoms would help to decide against ventricular hemorrhage. Owing to the rigidity present, some cases show a resemblance to tetanus, but the coma or other conditions of impaired consciousness and mentality would contraindicate this diagnosis. A careful study of the symptoms of the chronic affections given above, especially those which show the progressive character of the disease and its tendency to apoplectic exacerbations, will be sufficient to make the diagnosis, if, as is not often the case, the physician has in mind this usually diagnostically neglected disorder.

Pathology and Morbid Anatomy.—Two distinct views as to the origin of this disease have been held. As early as 1856, Virchow expressed the opinion that the primary condition is one of inflammation, which gives rise to a highly vascular neomembrane, from whose newly formed capillaries hemorrhages from time to time take place. The other view is that the hemorrhage is primary. It would seem certain that in some cases, at least, the hemorrhages take place independently of any inflammatory disease. Houssard, in 1817, was the first to show that the lesion was situated between the inner surface of the dura and what was then known as the outer surface of the arachnoid,—in other words, that it was a true dural disease. In a large majority of cases the lesions of this disease occur on both sides of the brain, very commonly in the same position, over a space which largely corresponds to the parietal bone and the motor region of the brain, often extending beyond these limits. As a rule, the appearance presented is that of layers of thin, soft tissue, which vary in color and consistency according to the age of the lesion. In recent cases, or in those in which recent hemorrhages have occurred, the blood may be

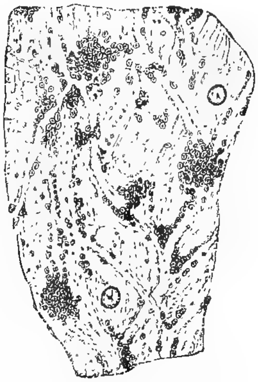
Fig. 204.



Pseudomembrane formed on the dura after a small hemorrhage. (Obersteiner.)

more or less fresh; older cases present firmer, denser, and lighter, but pigmented appearances. Sometimes several thin, flat sacs containing degenerated blood are found, one above the other. In rare instances abscesses are gradually formed. The convolutions of the brain beneath the position of the lesion, particularly if it is old and extensive, are flattened and atrophied. According to Wiglesworth, hematoma may be of venous origin, due to the impaired nutrition of the brain in atrophic and blood diseases. Obersteiner has described and illustrated the two processes by which the layers characteristic of this affection are formed. In Fig. 204 is shown the false membrane which is left after the absorption of the encapsulated blood resulting from a small hemorrhage from the dural veins into the subdural space, while Fig. 205 represents the organized new membrane which results from a condition of irritation of the inner surface of the dura, and which is probably, according to Obersteiner, accompanied with emigration of leucocytes out of this membrane. Such neomembranes may be deposited layer upon layer.

Fig. 205.



Neomembrane of the dura resulting from pachymeningitis hemorrhagica. (Obersteiner.)

Prognosis.—As the disease is essentially chronic and progressive, and occurs principally in the aged and broken down in health, the prognosis is unfavorable. Probably, if recognized early,—at the time that the membrane begins to form or when the first hemorrhage takes place,—something could be done by treatment, which should be for the relief of constitutional conditions, and perhaps in some instances the direct removal of the clot. When the false membrane has reached considerable dimensions, and after several acute apoplectic recurrences, the prognosis is unqualifiedly bad. A large hematoma may cause death rapidly.

Treatment.—If the disease is recognized during a hemorrhagic attack, treatment calculated to arrest the bleeding, as complete rest, moderate elevation of the head, and cold applications, is indicated. Blood may be abstracted by leeches to the temples or mastoid region. Venesection is commonly not to be resorted to, because of the weak condition of the patients. Diuretics, cathartics, mercury, opium, and bromides may prove of service. For the chronic conditions, attention to the habits of the patient, particularly the interdiction of alcohol, and the careful management of the kidneys, heart, and lung complications, are necessary. Remedies like the iodides and mercury may be tried as absorbents in the intervals between the attacks. Trephining for the removal of the clot has been employed in a few reported instances.

PURULENT INTERNAL PACHYMENINGITIS.

Purulent internal pachymeningitis, or suppurative inflammation of the internal surface of the dura, is extremely rare as an independent affection, but its existence has been noted. Its symptomatology is probably that of a subacute stage of hemorrhagic internal pachymeningitis; in other words, the phenomena are those of dural and pial irritation and brain compression, such as headache, vertigo, vomiting, septic fever, drowsiness, pressure paresis or paralysis, spasms, rigidity, and pupillary changes. Its causes are those of suppurative meningitis of other forms, as aural and nasal disease, purulent pleurisy, endometritis or peritonitis, ulcerative endocarditis, carbuncle, cranial injuries, and erysipelas. Commonly purulent pachymeningitis is associated with suppurative leptomeningitis, under which head its pathogenesis will be considered. In the case of facial erysipelas referred to on page 262 a careful autopsy was made by Drs. Osler, Jamieson, and the writer. As the intracranial lesion was mostly a widespread purulent internal pachymeningitis, and as records of such cases are rare, the notes made at the time of the autopsy will be given in some detail, and will serve as a description of the gross pathology of this disease. On removal of the skull cap, the external surface of the dura was found to be normal. Pacchionian granulations were numerous and distinct. In the longitudinal sinus was a recent clot. The inner surface of the dura of the right hemisphere was smooth; on the left side over the bases of the frontal lobe and the operculum it showed a grayish semipurulent exudation. The membrane here was closely adherent in a patch nearly two inches in diameter. Over a greater part of the anterior half of the hemisphere the dura presented a thin, recent, fibrinous exudation, with many localized spots of extravasation. Corresponding to the patch of solid exudate was an area of thick purulent lymph, covering the pia-arachnoid. The basal dura showed in the anterior fossa recent thin exudation with numerous ecchymoses. In the left middle fossa the dura was covered with a tenacious, distinctly creamy lymph. Stripping the dura from this fossa, an extensive layer of purulent exudation was found beneath it. It was here much more abundant than in the other regions. The longitudinal sinus and the right lateral sinus were free from exudation or signs of inflammation. The left lateral sinus along the attachment of the tentorium was closed by a thrombus. This was loose at the petrous portion where it turned downward and inward. The sinus was completely occluded and contained pus. The fifth nerve and the Gasserian ganglion were partly infiltrated with pus, but it did not extend into the orbit or along the course of the optic nerve. No suppuration was present in the sphenoidal cells. On the right side was also purulent exudation. The left tympanic mucous membrane

was swollen and hemorrhagic, but did not contain pus. The brain showed extensive subarachnoid gelatinous edema of the cortical sulci. The membranes and vessels at the base were normal. Over the anterior margin of the left temporal lobe, extending into the Sylvian fissure and over the posterior portion of the third left frontal convolution, was a layer of thick, curdy, purulent lymph lying upon the arachnopia. At the apex of the temporal lobe in the third temporal convolution was a very small purulent focus. When the Sylvian fissure was fully opened, it was seen that the exudation extended for a short distance into it. Sections of the hemispheres showed no region of focal disease. The lungs were edematous and congested; the left presented several septic infarcts, one at the anterior margin containing creamy pus. The spleen was large and contained no infarct. The diagnosis of purulent internal pachymeningitis must be based largely on a study of causation and the symptoms of pressure, irritation, and sepsis above given. The prognosis is unfavorable. Surgical treatment in rare instances affords some hope. Otherwise the treatment is that of suppurative leptomeningitis, which will presently be considered.

SEROUS INTERNAL PACHYMENINGITIS.

Under the name of serous pachymeningitis, Henoch, Gowers, and others have described a rare condition met with in young children and occasionally in general paralytics. This affection is to be distinguished from the serous meningitis in which the effusions are either cortical or ventricular, and which has been best described by Quincke; but both diseases originate from the same general pathological causes. In serous pachymeningitis, as described by Gowers, a membranous layer lines the dura and is continuous at the base with another layer covering the arachnoid, from which it is separated by a fluid. Such cases have sometimes been described as *external hydrocephalus.* The microscope may reveal extravasations in the outer layer. Enlargement of the head is present in some cases and absent in others. When, as may be the case, the brain is compressed and atrophied, the patient may show decided evidences of mental deficiency, but commonly cerebral symptoms are few in number. Redness of the scalp has been noted. Fever of irregular type may be present. Splenic enlargement has been found in children.

DURAL HEMORRHAGE.

Varieties.—Under the name of *meningeal hemorrhage* several varieties of hemorrhage of the dura are described; the chief of these are known as the *supradural* or *epidural* and the *subdural.* These affections are usually traumatic, and are of great surgical importance.

Supradural Hemorrhage.—When the extravasation is supradural the symptoms are chiefly general. Contralateral paralysis, however, may serve as a broad localizing indication when the bleeding is over the motor area, if external appearances are wanting. Unilateral affection of the pupil is often a sign of the utmost importance, particularly if one pupil is found widely dilated, the other being natural or contracted in size, and if the dilatation be on the side of the face corresponding to the injured side of the head. Jonathan Hutchinson has studied and discussed the importance of this valuable symptom, and in honor of him Jacobson proposed to call it the "Hutchinson pupil." Hutchinson regards the symptoms as due to direct or indirect compression of the third nerve. The pupils also furnish valuable indications as to the probability of recovery. The more dilated, insensitive, and immovable they are, the less favorable is the prognosis.

FIG. 206.

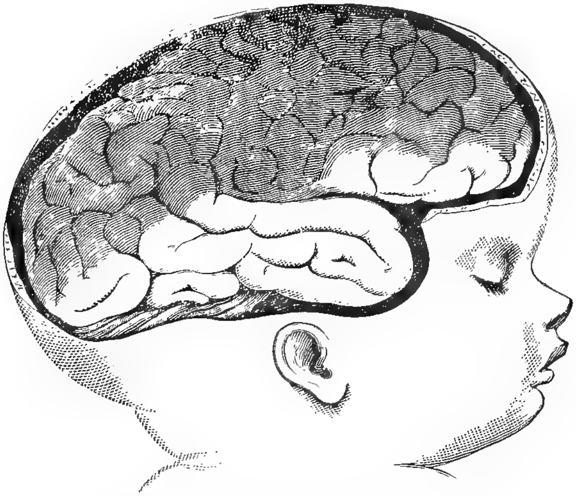
Sites of the most common varieties of meningeal hemorrhages (from the middle meningeal or medidural artery): *I*, hematoma, frontotemporal; *II*, hematoma, temporoparietal; *III*, hematoma, parietooccipital. (Elliot, after Krönlein.)

Subdural Hemorrhage.—Subdural or intermeningeal hemorrhage, if extensive, gives general symptoms much like those which are present in supradural clot,—namely, loss of consciousness, changes in temperature, in pulse, and in respiration, and vomiting. A subdural clot will usually bruise and possibly will even tear the brain surface. Cortical as well as dural spasm may be present. Para-

lytic symptoms will be definite and pronounced, as the lesion is in the motor region. Cheyne-Stokes breathing may or may not be present.

Mode of Origin and Treatment of Dural Hemorrhages.—Usually both supradural and subdural hemorrhage are due to serious injury; frequently fracture and hemorrhage are associated, but it is of vital importance to remember that the hemorrhage may occur without fracture, or that the latter may be confined to the internal table of the skull. As a rule, dural hemorrhages, traumatic or of other origin, are from lesions of the middle meningeal (medidural) artery. According to Krönlein, they may be divided, as shown in Fig. 206, into three classes, (1) frontotemporal, (2) temporoparietal, and (3) parietooccipital, according to the branches of the middle meningeal artery ruptured. The meningeal hemorrhages which so often give rise to the cerebral palsies of children, like those in the adult, are also commonly from branches of the meningeal arteries, and are therefore supradural or subdural, the latter giving rise to the more serious results. (Fig. 207.) In the newborn, or in very

FIG. 207.



Meningeal hemorrhage in a newborn child. (McNutt.)

young children, the usual symptoms of dural hemorrhage may be overlooked, and be noticed only when the child attempts to move its limbs or to talk. Most cases of meningeal hemorrhage due to indirect violence are subdural. The treatment of supradural and of subdural hemorrhage is mainly surgical, and in this field many brilliant results have been achieved, particularly in recent cases. The concurrent testimony of neurologists and surgeons is in favor of early trephining for the removal of the clot.

DURAL TUMORS.

Very many intracranial tumors grow from the pia or from the agglutinated inner and outer membranes, but occasionally neoplasms are limited to the dura in their development and give rise to symptoms which can be referred almost exclusively to this membrane. Such a case has been put on record by me, and a summary of it will serve to present the usual features of this affection. The patient, a man, thirty-two years old, when admitted to the hospital suffered from severe headache, sleeplessness, obstinate constipation, and loss of appetite and flesh. No paralytic symptoms were present, and the tendon phenomena were normal. The right pupil was slightly larger and responded to light more slowly than the left. The head pain, located by the patient over the right eye, was of varying intensity, and was always worse at night. In a short time the patient died with uremic symptoms. Eight years before his death he had been rendered unconscious by a blow on the head, and after this he suffered for a long time from headache, from which he eventually recovered, remaining free from the symptom until three months before his death. He used alcohol to excess. The autopsy showed an area of thickening of the skull about an inch and a half in diameter, beginning one inch above the line of the external meatus, being at this point strongly adherent to a tumor or thickening of the dura. The mass was flat, tough, almost circular, one eighth of an inch in thickness, and whitish yellow in appearance. The dura and pia were agglutinated and adherent to the brain beneath the growth, and both skull and brain were bruised and eroded. The growth was so situated as to cross the fissure of Sylvius over portions of the postcentral, subparietal, and supertemporal convolutions, its anterior limit being the middle of the latter. The lesion was strictly meningeal, but in enlarging had slightly invaded both the skull and the brain substance. Subsequent microscopical examination showed the growth to be syphilitic. Dural tumors are most frequently of traumatic origin. A fibroma may originate in this membrane as the result of a recent or an old injury, or syphilitic infection may be focussed in this way, as in the case just given. Carcinomata, especially the melanotic variety, sarcomata, the psammomata or small calcareous growths, and tuberculous growths may also arise from this membrane. The diagnosis of a tumor limited to the dura is not readily made, which is unfortunate, as such a neoplasm is very amenable to surgical treatment. It might be regarded as fairly determined in a case with a history of injury to the skull giving a symptomatology of severe localized pain, with tenderness on percussion in the same area, and with attacks also of vertigo and nausea or vomiting, spasms of dural type, and unilateral disturbance of the pupil. The affection might be confounded with a localized pachy-

meningitis, or leptomeningitis with various toxemias. The diagnosis of dural tumor was made in a case which came under my observation, and which proved to be chronic nephritis, with vascular and other changes and uremic phenomena. The prognosis is usually bad. Some of the growths can be successfully removed by trephining, and the syphilomata are amenable to active treatment with mercury and iodides.

DURAL ABSCESSES.

As the result of cranial injury or of infection in any of the various ways already referred to in speaking of purulent pachymeningitis, a more or less localized supradural or subdural abscess may be formed; but this affection need not be referred to in this connection further than to say that its diagnosis and treatment are sufficiently considered in the discussion of suppurative meningitis and under intracranial localization and abscess of the brain.

DURAL EPILEPSIES.

Forms of epilepsy dependent upon dural irritation need to be differentiated from cortical and other forms of convulsive seizure, but this subject will be more fully discussed under epilepsy, and attention is directed to it here merely in order to complete the survey of affections in which the dura plays the chief rôle. Experiments upon the lower animals, and especially those made by Dupuy, Brown-Séquard, Burdon Sanderson, Duret, and François Franck, and in 1874 by a committee of the New York Society of Neurology and Electrology, have shown that electrization of the dura produces muscular twitching, usually on the side of the body to which the irritation is applied. During an operation I have seen severe general spasms produced by a faradic current accidentally applied to the dura. I have also recorded cases in which spicules of bone in the dura gave rise to serious chronic epileptiform attacks. A distinct separation cannot always be made between dural and cortical epilepsy; but in the latter the convulsion uniformly begins in the side opposite to the half of the brain that is irritated. In dural epilepsy, and perhaps in other forms of spasm due to trigeminal irritation, the attack frequently begins on the same side as the focus of irritation, but unfortunately this distinction is not radical, as the convulsions may begin on either side. Definite signal or initial symptoms are usually wanting in dural cases, one-half of the body instantly plunging into spasms.

LEPTOMENINGEAL HYPEREMIA AND ANEMIA.

Cerebral anemia, or congestion of the brain, has always been a favorite diagnosis with some practitioners; and yet the occurrence of the disease as an independent affection has been doubted. It has a real existence, but is not so frequent as was once supposed.

Hyperemia of the brain, for anatomical reasons, is largely hyperemia of its soft membranes—the arachnopia. The pial vessels ramify everywhere over the surface of the brain, covering the crests of the convolutions, dipping into the fissures and threading their intricate windings and branchings, and, entering the cortex, passing as nearly straight vessels into the interior of the brain. Even the central or ganglionic system first takes its way through the membranes in certain areas at the base of the brain. Membranes and brain substance take part together in congestion, as in anemia. All encephalic anemia or hyperemia is, therefore, also meningeal—that is, meningo-encephalic—and chiefly cortical, as the arteries become smaller and practically do not anastomose in the interior of the brain. In the light of these facts, and as the varieties, clinical history, etiology, pathology, diagnosis, prognosis, and treatment of hyperemia and anemia of the membranes cannot be separated from those of the brain substance, they will be considered under diseases of the brain itself.

EXTERNAL LEPTOMENINGITIS (so-called ARACHNITIS).

Some of the older writers describe arachnitis as an independent disease; others confuse under that name varieties of what are now known as leptomeningitis and pachymeningitis. Certainly a pure arachnitis cannot be recognized through a definite symptomatology. If, as has been suggested, the arachnoid is not regarded as a separate membrane, but simply as the parietal layer of the pia, then an inflammatory affection of this membrane might perhaps be better spoken of as an *external leptomeningitis*. L. Meyer found epithelial products on the superior surface of this layer or membrane in chronic cerebral irritation. The enlargements and increase in the Pacchionian granulations in drinkers, in epileptics, and in the insane may perhaps be regarded as disease of this layer. In newly born infants hemorrhages of the pia or brain cause imbibition of blood by the so-called arachnoid; and opacities and thickenings from repeated congestion are usually met with in old age. In a large number of autopsies made in Vienna upon suicides, considerable arachnoidal hypertrophy was noted. (Rosenthal.) Small disseminated white patches of thickened arachnoid may be scattered over the whole brain convexity, especially in idiots. (Obersteiner.)

LEPTOMENINGITIS.

Method of Discussion, and Varieties.—How best to discuss leptomeningitis is a question that confronts the medical teacher and writer. Authors differ much in their ways of handling the subject. Some, like Huguenin, take many pages for the separate consideration of various forms of the disease. A common method of recent textbooks is to subdivide it into purulent or suppurative leptomeningitis, tubercular leptomeningitis, and epidemic cere-

brospinal meningitis. Some treat the entire subject under one head, not even excepting the epidemic cerebrospinal form. All forms of encephalic leptomeningitis will be included here in one general class, special paragraphs being devoted to the clinical history of particular etiological varieties, when these are stamped with peculiarities allowing them to be differentiated. In the first place, *acute* and *chronic* leptomeningitis or inflammation of the arachnopia must be recognized. Acute leptomeningitis includes a number of special varieties, as *purulent* or *suppurative*, *non-purulent*, usually infantile, *serous*, *tubercular*, and *epidemic cerebrospinal*. The symptomatology is more determined by localization than by the pathology or etiology of the disease, and for this reason it is advisable to have especial regard to the situation and diffusion of the lesions. Leptomeningitis, therefore, from this point of view, may be *basilar*, when the base of the brain is solely or chiefly affected; *verticalar* (Barr), when the convexity is the region chiefly invaded; or *general*, when all the surfaces of the brain are attacked with some approach to uniformity. Leptomeningitis either of the base or of the convexity may also show a tendency to occur with most severity in special localities, as in the cervico-oblongatal region, or in and around the Sylvian fossa. The term *idiopathic* is sometimes applied to forms of meningitis, but it should be discarded. Here, as elsewhere, the term is generally a cloak for ignorance. Many etiological varieties of leptomeningitis might be erected, but it is preferable to consider these, with the exception of the tubercular and syphilitic forms, under general etiology.

General Symptoms Common to all Types of Acute Leptomeningitis.—All forms of intracranial leptomeningitis may have certain symptoms, both general and local,—the former dependent upon the fact that the brain meninges are involved, and the latter upon the special meningocortical areas which are invaded. According to the etiological and other varieties of the meningitis, however, the symptoms will differ somewhat, but always within a certain range. All forms of pial inflammation give rise to headache, and this, on the whole, is one of the most constant phenomena; but even this well accredited symptom is not necessarily present in every case; certainly it is not present in every stage of the disease. Its presence and severity are probably dependent upon the extent of the lesion, and sometimes upon its situation. The pia and brain substance, unlike the dura, are poorly, if at all, supplied by sensory nerves. When both the dura and the pia are attacked with inflammation, or when the Gasserian ganglion and the fifth nerve at the base are involved, as they frequently are, pain in the head is likely to be of great severity. Great increase of intracranial tension probably also gives rise to pain. In a case seen by me with Dr. W. H. Rihl, of Philadelphia, the patient being a man twenty-eight years old, no

headache was present during his illness, which lasted several weeks; the autopsy showed disease over a considerable area of the motor convexity and the mesal surface of the hemisphere. I have made autopsies in several other cases of convexity meningitis in which no history of headache was present. The truth about headache and localized head pains may be summed up in the statement that they are symptoms of frequent but not of constant occurrence. Pain and hyperesthesia of the head and face and of other parts of the body may be produced by irritation of cortical sensory areas. Peculiar mental changes are common, and are sometimes determined by the special regions involved, as the prefrontal lobe; but they are more frequently due to general irritation and pressure. They may be irritability, peevishness, restlessness, delirium, or maniacal excitement, or, on the other hand, depression, moodiness, obtuseness, somnolence, apathy, stupor, or coma. These symptoms of disturbed mental action differ much in different stages of the disease. In the prodromal periods of protracted cases the irritative symptoms predominate; in the middle and later stages, those of depression. Optic neuritis, either single or double, may also be present, but it may be absent in any type. Vertigo is a symptom in a fair percentage of cases. Nausea and vomiting, like vertigo, may be present or absent in leptomeningitis, although these are rather constant phenomena in acute inflammations of the dura. Changes in temperature, pulse, and respiration belong to all types of acute leptomeningitis; but they differ much in character in different varieties, and even in different cases of the same variety. The temperature may be greatly or moderately elevated, or even sometimes depressed. In one of my cases of tubercular leptomeningitis in a child, the temperature during the entire period of the disease ranged between 97° F. and 99.7° F., but I have known it to range throughout between 101.2° F. and 107° F. The pulse may be slow, irregular, and intermittent, or very frequent; it seldom becomes wiry. Respiration is often strikingly affected, Cheyne-Stokes breathing being common as the disease progresses to a fatal termination.

Symptoms of Basilar Leptomeningitis.—Any of the pathological or etiological varieties of leptomeningitis may be limited to the base of the brain. The symptoms presented by a case of acute basilar leptomeningitis will depend upon its diffusion and intensity. When severe and widespread, all or nearly all of the cranial nerves may be involved, giving affections of smell, sight, hearing, and taste, various forms of strabismus from paralysis of the ocular muscles, or facial, trigeminal, spinal accessory, or lingual paralysis. Spastic affections of the muscles supplied by any of the motor cranial nerves and affections of any of the sensory cranial nerves may also be present. Occasionally a severe trifacial neuralgia is due to a localized meningitis at the base of the brain and in the vicinity of the

Gasserian ganglion. The ocular and facial muscles are specially likely to suffer, because of the tendency to concentration of the inflammation near the origin of the nerves which supply these muscles. Affections of taste and smell may be due to the spreading of purulent inflammation to the cortical olfactory tracts and centres, as well as to involvement of these nerves of special sense. Temperature changes may be present, due to invasion of heat centres in the pons and in the vicinity of the tuber, and polypnea or rapid breathing may be a symptom from involvement of the latter region. (Ott.) Forms of alternating hemiplegia or hemiparesis may be caused by implication of the cranial nerves on one side and pressure on the motor tracts supplying the limbs of the other half of the body. Optic neuritis is a frequent symptom, and deafness with signs of auditory neuritis is infrequent. Retraction of the head and tetanoid symptoms may be present in almost any case, but are more commonly found when the region of the oblongata is specially attacked. Gee and Barlow have described a form of basal meningitis which usually occurs in children under two years old, and which has special features giving it diagnostic importance. It is probably, at times at least, tubercular. The most common symptom is cervical opisthotonos; other symptoms, also common to different varieties of leptomeningitis, are fever, vomiting, rigidity, local or general spasm, and hydrocephalus, which is supposed to be due to the closure of the ventricular cavities by the gluing together of the cerebellum and the oblongata.

Symptoms of Leptomeningitis of the Convexity.—Any of the varieties of leptomeningitis may attack the convex surface as well as the base of the brain. In convexity or verticalar meningitis cranial nerve symptoms will of course be absent, unless the process at the same time attacks a spot here and there at the base, as sometimes happens. As inflammation occurs most frequently over the motor cortex, the predominating symptoms will be motor, such as paresis or paralysis of face or limbs, motor aphasia, usually partial, and spasms, often unilateral, or beginning unilaterally and becoming general, and showing themselves clearly to be of cortical type. Occasionally the primary cortical visual centres are involved, giving hemianopsia or sector defects, or various irritative visual phenomena, such as phosphenes, color vision, and hallucinations of sight. Word blindness and letter blindness, usually partial, are observed in rare cases, as are also word hearing defects or irritative auditory phenomena. Mental symptoms are nearly always present, and will vary with the intensity of the lesion in special localities. When the prefrontal lobes are specially affected, hebetude, inattention, moroseness, and apathy may be present. Delirium is a more frequent and pronounced symptom than in the basilar form, while optic neuritis is decidedly rarer.

Acute Purulent Leptomeningitis.—The statement has been made that all leptomeningitis is purulent, but in serous meningitis pus is not present, at least not to the naked eye. It is probable that occasionally a true leptomeningitis does not pass beyond the early nonsuppurative stage, the disease aborting or the patient succumbing to the toxic influence, which at the same time that it initiated a meningeal inflammatory process overwhelmed the central nervous system. This may be the explanation of those cases which present during life striking symptoms of meningitis, but which show slight or no evidences of inflammation of the membranes after death. Some forms of acute delirious mania have been supposed, on rather uncertain grounds, to be due to a rapidly developed leptomeningitis, but it is more probable that these cases are dependent upon a general toxemia which affects the entire brain and perhaps in some instances the entire nervous system. The general symptoms which have been given as common to all types of leptomeningitis, with the paragraphs describing the disease of the convexity and of the base, may be regarded as a clinical picture of an average case of purulent leptomeningitis. It only needs to be said that in the purulent as in other forms of leptomeningitis great variations in symptoms are observed. It may begin abruptly and run a rapid course to death. This is particularly true in cases associated with abscesses of the brain or with extensive suppurative disease of the cranial bones. As a rule, the affection is more or less prolonged, and in rare instances but a few symptoms are present. Latent cases may depend upon the fact that the greatest virulence of the disease is not expended upon regions that give rise to active symptoms, but on more latent cerebral zones, as the prefrontal or the right temporal lobes, or the cerebellar hemispheres.

Serous Meningitis.—Under the name of serous meningitis (*meningitis serosa*) Quincke has described an affection which he believes to occur frequently, which is to be distinguished from all the other forms of meningeal inflammation. The inflammation of the membrane results in serous effusions, both cortical and ventricular. Some cases come on rapidly, some slowly; some are acute, others chronic. Fever is usually slight, or may be absent or unnoticed. When present it is of irregular type. Head pain and retraction of the neck are not infrequently absent. Mental changes may occur, but are not commonly so decided in character as in other types of leptomeningitis. Other symptoms often present are insomnia, joint pains, delirium, local palsies, vomiting, slow and irregular pulse; in rare cases there are unequal pupils, epileptiform convulsions, and optic neuritis with secondary atrophy. The disease is one of childhood and youth. The diagnosis of the acute form is to be made chiefly from suppurative and tubercular meningitis, which can be done by close comparison with the symptomatology of these affections.

From suppurative meningitis two of the most important diagnostic distinctions are the less marked fever and the more frequent presence of choked disks. In the serous form the symptomatology is of milder type and more variable in character. It is almost impossible in some cases to distinguish the disease from tubercular meningitis. In serous meningitis the temperature is practically normal. Huguenin treats at length of an acute nontubercular infantile leptomeningitis, an affection which has been described as acute nontubercular hydrocephalus. He believes the hydrocephalus to be of inflammatory or at least of hyperemic origin. Quincke's description of serous meningitis applies in some particulars to this form of infantile leptomeningitis, which may therefore be regarded as one of the forms of serous meningitis. Effusions may occur in these cases in a variety of ways, as by fluxions to the brain and pia; in connection with general dropsy, particularly in such diseases as scarlet fever; in the so-called marasmic disorders; with neoplasms, abscesses, and other focal lesions; and in venous stases from disease of the lungs or of the heart, and with obstructive deformities. This affection may come on in children previously healthy, or it may follow infectious diseases, diarrhea, injury, or any depressing or deteriorating influences. In many of the cases examined post mortem no macroscopic purulent appearances have been found. The symptomatology differs but little, and in some cases not at all, from that of tubercular leptomeningitis. A prodromic or at least a long prodromic period is not as likely to occur as in the tubercular form. In rare instances the disease is fulminant, arising with great suddenness and violence, as with severe general convulsions. The first stage is not long; slight fever may be present at the onset; severe convulsions are somewhat common; headache, restlessness, moaning, delirium, moroseness, vomiting, constipation, twitchings, hyperesthesia, photophobia, and sensitiveness to sounds are frequent. The symptoms differ somewhat according to the age of the child. Headache will be complained of by older children, who also early in the disease may not be able to stand or walk. Spasm of the glottis may be present. Sometimes the patient dies in a few days after general convulsions. If the child survives such convulsive attacks, the symptoms above described will be generally aggravated, and others will be added, as the hydrocephalic cry and abnormal reactions of the pupils. The pulse, respiration, and temperature may show great variations; a pulse abnormally slow may rise in frequency with great rapidity, and the same is practically true of temperature and respiration. Vomiting may be severe and persistent. As a rule, the patient sinks gradually into coma, but occasionally recovery takes place. This may not be perfect, the commonest symptom which remains being hydrocephalus. The duration is variable, death coming on in some cases in a few hours or a day, but ordi-

narily the disease lasts from ten to thirty days. While the above is a sketch of the disease as it usually occurs, the symptoms, as in tubercular leptomeningitis, may differ widely from this picture.

Acute Tubercular Leptomeningitis.—Tubercular meningitis as it occurs in children is one of the best known intracranial diseases. In some textbooks the descriptions would seem to indicate that it is invariably a disease of childhood; but it may occur at any age, in either acute or chronic form. In one of my cases, a man sixty-five years old, who had an internal pachymeningitis of one hemisphere, miliary tubercles were scattered over the postfrontal and parietal lobes of the other hemispheres, and there were also several foci of superficial softening caused by tubercular obliterations of small vessels. Both lungs were infiltrated with miliary tubercles. Similar cases in the aged are seen with comparative frequency in the postmortem examinations made at the Philadelphia Hospital. Cases of tubercular leptomeningitis verified by autopsies have been observed by me at all ages from a few months to seventy years. Relatively, however, the disease is less common after middle life. Experience would seem to confirm the view, long held, that the form of tubercular meningitis which occurs in early childhood is usually basilar, or rather that the lesions largely predominate at the base of the brain. It is customary to study acute and subacute tubercular meningitis as occurring in more or less definite stages; but, while this has some practical advantages and is borne out to a fair degree by the facts, these stages frequently cannot be separated. One of these subdivisions, which can bring the greatest number of clinical facts to its support, is into (1) a prodromal or invasional period; (2) a period of irritation; (3) a period of depression; and (4) a terminal period. Invasion symptoms are emaciation, gastro-intestinal disturbances, such as anorexia, nausea, vomiting, diarrhea or constipation, vertigo, fever, usually of moderate degree, sleeplessness or unusual drowsiness, irritability, restlessness, peevishness, languor, apathy, aphasia, general convulsions, and hysterical attacks, although the last are not common. In the early period or irritative stage of the fully developed disease the most common symptoms are headache, fever of irregular type, constipation, *tache cérébrale*, local spasms or general convulsions, paresis or paralysis of cranial nerves, particularly of the ocular and facial nerves, sometimes paralysis of the extremities, and spastic phenomena varying according as the motor convexity or the base is involved. Besides the *tache cérébrale*, or *tache méningitique*, which is a red line made by passing the finger over the skin, various cutaneous eruptions are often observed, as erythema, herpes labialis, and urticaria-like eruptions. In one case seen by me, a peculiar odor was given off like that from the cutaneous glands. Other symptoms are flushings, retraction of the head and neck, and opisthotonos; sinking of the abdomen, occa-

sionally followed by tympanites; nodding, side to side, and rotary movements of the head; nystagmus and pupillary changes, more commonly dilatation, but sometimes contraction, and often differences on the two sides; and occasionally great thirst. Deep seated or superficial tenderness may be noticed, and this, which is also sometimes present in cases of epidemic cerebrospinal meningitis, may be due to the concurrence of neuritis with the meningitis. In one case pressure along the femur seemed to cause much pain. Coldness in the extremities and difficulty in swallowing are occasionally observed. The symptoms just given are those of what might be termed the fully formed disease in its usual type, and include those of the period of irritation and, in part, those of the stage of depression. In the fourth or terminal period, many of the symptoms already described are added to or become intensified or aggravated; paresis deepens into paralysis; local or general spasms occur with greater frequency; indifference becomes apathy, and apathy stupor; respirations become irregular, and finally breathing is of the Cheyne-Stokes variety; emaciation is extreme, evacuations are involuntary, and temperature changes, like those of the pulse, are very marked.

Tubercular Cerebrospinal Meningitis.—Cerebrospinal meningitis is occasionally of tubercular origin and has some clinical interest. Cases have been put on record by Magnan, Hayem, Liouville, Shaw, Moxon, Debove, Galliaux, and Eskridge. The case recorded by Eskridge was seen and studied by the writer in consultation. The post mortem and the microscopical examination made by Dr. L. Brewer Hall showed tubercles present chiefly in the membranes of the spinal cord, less abundantly in the meninges of the brain, and in slight degree in the lungs. The bodies and horns of the ventricles were enormously dilated. Although tubercular deposits were revealed by the microscope, they were not detected by macroscopic examination. Usually in cerebrospinal meningitis the deposit takes place first and is most abundant in the membranes of the brain, but in rare instances, as in the above case, this order of events is reversed. Debove has also recorded a case in which the primary lesion was spinal, the patient being a man twenty-nine years old, who was suffering from pulmonary phthisis. The point has been made that cases of this kind should not be classed as tubercular cerebrospinal meningitis, but rather as instances of general tuberculosis, with accidental or special tubercular deposits occurring in the encephalic and spinal membranes; but such a criticism has little force, as in most cases of tubercular meningitis some involvement of other organs will be found. The etiology, pathology, prognosis, and treatment of tubercular cerebrospinal meningitis are practically the same as those of tubercular meningitis of any form. The symptomatology will vary according to the absolute and relative extent and severity of the lesions which exist within the spine and within

the cranium. In the case reported by Eskridge, the patient, sixteen months old, was taken sick with fever, headache, tossing of the head from side to side, with a temperature of 103° F., pulse 150, respiration 84, and tetanic convulsions, with some imposed clonic movements and rigidity of the limbs. The fever was decidedly paroxysmal in type. Cheyne-Stokes respiration was sometimes present. Retraction of the head and choreiform movements of the head and neck were prominent symptoms. The child was at times passionate and almost maniacal. For short intervals it was more conscious and rational and suffered much less than during the ordinary course of the affection. Stomach irritability and diarrhea were present. Late in the disease the child suffered terribly with head pains, and had severe attacks of convulsions with opisthotonos, and irregular clonic spasms, rigidity, and contractures. The duration of the case was eight months. Some of the most important conclusions drawn by Eskridge are, that tubercular deposit may first take place in the meninges of the cord, and then extend to those of the brain, although the reverse is the rule; that tubercular cerebrospinal meningitis gives rise to special symptoms, and by a careful analysis of a number of cases it may be recognized; and that it is also probable that many cases of so-called cerebrospinal meningitis that have a duration of several months and then prove fatal are tubercular in character.

FIG. 208.

Hydrocephalus in a case of subacute tubercular meningitis.

Acute Hydrocephalus.—It will be seen from the preceding paragraphs that one of the commonest results of acute meningitis of various types is effusion into the ventricles, with great distention of these chambers, while effusion beneath the arachnopia also occurs, but with less frequency. This gives rise to hydrocephalus or dropsy of the brain, which is called internal when ventricular, and external when subpial. The term *acute hydrocephalus* has been somewhat confusingly employed in textbooks. Sometimes it is discussed as the name of a special affection; often it is used as a synonym for tubercular meningitis. Both of these usages may lead to error, as hydrocephalus is always a result of some pathological process, and as tubercular meningitis may be present without hydrocephalus. Sometimes even

in the acute cases hydrocephalus is in part or altogether mechanical, as when the foramen of Magendie is closed by adhesion between the cerebellum and the oblongata, in Gee and Barlow's oblongatal form of meningitis. It is supposed that in rare cases an inflammation of the ependyma or lining membrane of the ventricles, a so called *ependymitis*, gives rise to ventricular effusion. The appearance of the head and face in a case of hydrocephalus associated with subacute tubercular meningitis is shown in Fig. 208. Dr. Robert Whytt, of Edinburgh, in 1768, in describing the most common form of acute hydrocephalus, first directed attention to the connection of this affection with acute inflammation of the meninges. Later it was shown that the membranes were usually the seat of tubercular deposits.

Epidemic Cerebrospinal Meningitis.—Usually in works on neurology and general medicine cerebrospinal meningitis is discussed separately; but, while this plan has its advantages, no good reason exists for not considering the disease under the general head of meningitis or leptomeningitis, except its special importance as an endemic or an epidemic. Its lesions are similar to those of suppurative meningitis of other types, and its pathogenesis shows the presence of the same microorganisms, although some of these, and especially the pneumococcus, play a predominant part in epidemic cerebrospinal meningitis. This disease has been known in an endemic or an epidemic form for centuries, although in former times, according to Stillé, it was generally confounded with typhus. It was not recognized as a special disease until the beginning of the present

Fig. 209.

Convulsion in a case of cerebrospinal meningitis. (J. Lewis Smith.)

century; its literature has since become voluminous, and the bacteriological investigations of recent years have given a new stimulus to its study. While usually endemic or epidemic, it occasionally occurs sporadically. As Stillé says, no other disease wears

such various masks of symptoms. Marked prodromes are often absent, but when present they are such as general malaise, headache, and pains in the limbs. These may last an hour or two, or in rarer instances from one to two weeks. Occasionally neck pains and nausea or vomiting precede the full outbreak. The symptomatology of acute cerebrospinal meningitis is more like that of the purulent than like that of the tubercular form; but its symptoms may simulate closely those of any other form of leptomeningitis, and its diagnosis needs to be made from a study of its methods of onset, the presence of an endemic or an epidemic, and the facts which show the involvement of both the cerebral and the spinal meninges. The onset varies; but a typical case is usually ushered in by a chill, or at least by a chilly sensation, violent headache and vomiting being present from a very early period. Delirium is soon marked; fever rapidly develops, and is of various types, intermittent, remittent, or continuous. The temperature may cover a wide range, perhaps reaching an elevation of 106° or 107° F.; pulse and respiration also vary considerably, the former being usually quickened. Of the above symptoms headache, vertigo, and delirium, like the fever, may show great irregularities. As the base of the brain is the seat of frequent and severe lesions, cranial nerve symptoms, such as anosmia, paralysis of the ocular muscles, facial paresis or paralysis, inequality of the pupils, deafness and disturbances of articulation, hyperesthesia and anesthesia of parts of the face or head, are frequent. The affections of the motor cranial nerves may be spastic as well as paralytic, giving facial twitchings, trismus, nystagmus, spastic strabismus, and spasmodic torticollis. Photophobia is common. Intraocular changes vary from passive congestion to optic neuritis. In rare instances thrombosis of the central retinal vein occurs. When the convexity is the seat of severe lesions the symptoms will be similar to those of verticalar leptomeningitis of any type,—hemiplegia or hemiparesis, monospasms or unilateral convulsions, cortical affections of sight, of hearing, or of speech, or special psychical disturbances, according to the centres involved. General convulsions occasionally usher in the disease, or they may appear during its course, as may also muscular and tendinous twitchings. Epistaxis was particularly noted by Flexner and Barker in the epidemic studied by them. Of cutaneous eruptions, herpes is the most common, and may be present on the lips and in other localities; petechial and purpuric eruptions are often present, and have given the disease one of its names, that of *spotted fever*, which is not a good designation, as the spots are absent in many cases. One of the commonest eruptions noted by Flexner and Barker was a purplish mottling which appeared and disappeared almost under the eye. Spinal symptoms are retraction of the head and neck, opisthotonos, rachialgia, spinal ten-

derness, spastic or paretic conditions of the limbs, retraction of the abdomen, paralysis of the bowels and bladder, and changes in the knee jerk, which is frequently absent or diminished. Contracture inflection, sometimes known as "Kernig's symptom," is often present, and is due, according to Friis, to an inflammatory affection of the nerve roots of the cauda equina. Hyperesthesia of the limbs is mentioned by some authors as among the spinal symptoms, but it is probably oftener due to the concurrence of multiple neuritis with the meningitis. It was first suggested by me that neuritis was probably present as a complication or a coincidence in some cases of cerebrospinal meningitis, and that in others the infection caused a neuritis rather than a cerebrospinal disease. Constipation is the rule, but diarrhea is occasionally noted. Albuminuria is frequently found, but polyuria and saccharine urine are not common. Flexner and Barker found leucocytes in the active stages of the disease. In the terminal period of fatal cases the patient usually develops stupor, coma, Cheyne-Stokes breathing, and great variations of temperature. Death is due to the profound blood changes, or to local mischief within the cranium. The disease may be extremely malignant, the patient succumbing within twenty-four to forty-eight hours. Many fatal cases die within two weeks; and those which recover often begin to improve before the expiration of the second week. The disease has been subdivided according to symptomatology and course into the *fulminating*, *acute*, *subacute*, *intermittent*, and *abortive*, terms which speak for themselves. Moderately frequent sequelæ are chronic headache, hydrocephalus, contractures, local palsies, and mental changes. Owing to the frequency of aural disease, such as otitis media, deafness is often a sequel, and this in young children may lead to deaf mutism. Pneumonia is one of the most frequent complications, and others are pericarditis, endocarditis, and affections of the joints. Other epidemic diseases, measles, scarlet fever, and mumps, may be more or less rife at the same time.

General Etiology.—The general etiology of leptomeningitis has a wide range. The disease may, in the first place, be due to certain constitutional or diathetic conditions, as syphilis, rheumatism or gout, and erysipelas. Other causes, either predisposing or provocative, are sunstroke, traumatisms, aural and nasal disease, alcoholism, nephritis, sclerosis of the bloodvessels, intracranial tumors, abscesses, aneurisms, embolisms, and hemorrhages. The vast majority of cases, if not all, are due to infection. They may arise in connection with almost any of the known infectious diseases, as with typhoid fever, scarlet fever, variola, diphtheria, or influenza, or in connection with almost any toxic agent, as lead, arsenic, mercury, copper, and carbonic oxide or other poisonous gases. Cases of so-called idiopathic leptomeningitis and those for which

no cause can be found are probably dependent upon undiscovered infection. That leptomeningitis, either cerebral, spinal, or cerebrospinal, occurs during the progress and decline of influenza, cannot be doubted in the light of the evidence which has been presented by various observers, and particularly during the epidemics of the last few years; but it is a rare concomitant or complication. Syphilis, while more commonly a cause of chronic leptomeningitis, occasionally gives rise to the acute form, sometimes in connection with gummata, or nodose periarteritis, and less frequently as an independent affection. It is important to recognize this mode of causation, because of the rapidity with which acute syphilitic cases may give way to treatment. Formerly under the head of metastatic meningitis were considered the varieties of leptomeningitis associated with pneumonia, ulcerative endocarditis, pyemia, pleuritis, pericarditis, dysentery, and a few other diseases. The term metastatic is no longer desirable. In coryza, pneumonia, and parotitis the same microorganisms have been found as in leptomeningitis. The infection is carried by the blood from the ordinary sites of these diseases to the membranes of the brain. Sunstroke, while more frequently the cause of pachymeningitis, may in rare cases give rise to inflammation of the arachnopia. Traumatism may act directly through contusions, lacerations, and fractures, or may exert an indirect influence by causing hemorrhagic extravasations. Suppurative disease of the mastoid cells, and of the middle or internal ear, with which sinus thrombosis may be associated, nasal disease, traumatic or other affections of the frontal sinuses and operations on these parts, diseases of the eye, orbit, mouth, or throat, may all be followed by leptomeningitis. Phlebitis originating in the nasal, buccal, or pharyngeal cavities may extend by anastomosing veins through foramina in the floor of the skull to the intracranial veins, sinuses, and membranes. Alveolar abscess and necrosis of the jaw, and facial and labial carbuncle, have been followed by inflammation of intracranial vessels and membranes. An intimate connection between the veins of the mouth, face, and pharynx and those of the cranium is by way of the foramina of Vesalius. The pterygoid plexus communicates freely with the facial veins and with the cavernous sinus through these foramina. Gowers, Fox, and others speak of a leptomeningitis due to overwork of the brain, but this is probably rare as an acute affection, although a chronic low grade leptomeningitis, as shown by Bevan Lewis and Batty Tuke, may doubtless be developed in this way. Occasionally cases which appear to be clearly instances of a meningeal inflammation occur in overworked and overworried school children, and in adults engaged in hard and embarrassing pursuits, but doubtless in most of these cases the explanation is to be sought in the reduction of the system, which allows an infection to do its work. The special causes

of serous meningitis, as given by Quincke, are acute and chronic alcoholism, injuries, excessive mental effort, and acute infectious diseases, although he does not believe in its bacterial origin. Tubercular meningitis is, of course, due to the presence of the tubercle bacillus, but many general reducing causes may lead to the development of the tuberculosis, as heredity, poor food, bad hygienic surroundings, and especially overcrowding. Epidemic cerebrospinal meningitis most frequently occurs in winter or spring, and, like other forms of meningeal inflammation, attacks those who are crowded together and are living in general under bad hygienic surroundings. It occurs in country districts, and here probably as much because of bad sanitation as for other reasons. The causative influence of climate and that of miasmatic conditions have not been established. The disease is probably not contagious, or, if contagious, is so to a slight degree only. Purulent meningitis may occur at any age, and the sexes show but little difference. Tubercular meningitis is most frequent in children and young adults, who are also most frequently the victims of cerebrospinal meningitis, which has occurred in children less than six months old; it may, however, attack persons of any age. Statistics with reference to sex are so uncertain that nothing positive can be said about this factor.

Pathogenesis.—The cerebrospinal membranes form a remarkably favorable breeding place for bacteria. (Putnam.) The microorganisms which give rise to purulent leptomeningitis are of several

FIG. 210.

Some of the microorganisms of leptomeningitis: *a*, staphylococcus pyogenes aureus; *b*, streptococcus pyogenes; *c*, pneumococcus or micrococcus lanceolatus; *d*, bacillus tuberculosis.

kinds, and include the streptococcus pyogenes, the staphylococcus pyogenes aureus, the intracellular diplococcus of Weichselbaum, and the pneumococcus or micrococcus lanceolatus. Tubercular leptomeningitis originates from the tubercle bacillus, which may find its way to the membranes of the brain from diseased glands, joints, and bones, lungs, intestines, or any other tissues or organs. All recent investigators are agreed that the pneumococcus is the microorganism most frequently present in epidemic cerebrospinal meningitis. It has been estimated that from eighty to a hundred per cent. of healthy people carry about in their mouths this microbe, a fact which makes it unnecessary to search for the cause of the disease in air, soil, or water. These micrococci are most frequently carried from the mouth

into the intestinal tract and thence into the blood current. (Flexner and Barker.) A moment's consideration will show that in many of the above cases septicemia is the real cause of the meningitis, as when it develops in connection with various purulent diseases, disorganizing clots or thrombi, caseating glands, endocarditis, or phlebitis. The symptoms in leptomeningitis are partly due to the exudation, partly to the irritation of the sensitive membranes by the micrococci, and partly to the absorption of their toxic products. (Putnam.)

Pathological Anatomy Common to all Types of Leptomeningitis.—The macroscopic appearances vary, in any form of leptomeningitis, with the stage of disease at which death occurs. As this fatal termination is usually after exudation and suppuration have taken place, the changes are, as a rule, of striking character. An inflammatory process may have been initiated in the arachnopia by the infection, death resulting very early from an overwhelming toxemia, and therefore few gross appearances are present, and these are usually those of intense hyperemia of the swollen tissues, varying from a widespread vivid redness to scattered patches of greater or less intensity of coloration. It may be correct to regard such cases as instances of inflammation without exudation, although the fatal result must not be attributed to the meningitis, but to the toxemia. Even in cases where cultures show the presence of the microorganisms which usually give rise to the meningitis, no gross appearances may be observed. In a case seen by me, the patient dying with symptoms of acute delirious mania, the membranes showed no macroscopic changes, but cultures made from the cerebrospinal fluid revealed the pneumococcus, and the staphylococcus aureus and albus. Certain appearances are common to all varieties except the pure serous form of the disease. These are areas of redness scattered over the surface, opacities most marked over the basal subarachnoid spaces, along great vessels, and over the fissures, and exudates of lymph and collections of pus here and there or diffused over very large surfaces. Occasionally, in old cases, caseation or some other form of tissue degeneration takes place. The exudate may be in points or in spots; more commonly it is in isolated or in imperfectly united areas of irregular shape. The velum interpositum and the choroid plexus may show decided changes, even of a purulent character. Pus, wherever found, varies from a white or whitish yellow to a greenish color. The ventricles are frequently but not invariably dilated, the dilatation which causes an acute hydrocephalus being more constant in the tubercular than in other varieties, except the serous meningitis of nontubercular origin. With less frequency the fluid in the spaces beneath the arachnopia is also increased. The fluid in either locality is not infrequently turbid from the presence of lymph. Occasionally an in-

flammatory or a granular appearance of the ependyma is observed. The appearances, even in advanced cases, vary, of course, with the localization and diffusion of the lesion. Verticalar or convexity leptomeningitis shows the most striking changes over the motor cortex and around the Sylvian fossa, but fibrinous exudate or pus may be seen anywhere on the lateral or mesal aspect of the hemicerebrum, or on the basal temporooccipital cortex. Meningitis occurring here probably should be classed with convexity meningitis rather than with the basilar form, which attacks chiefly the central regions of the base. In basilar leptomeningitis the opacities, deposits, exudates, and pus involve the various cranial nerves and vessels, and are often especially marked in the interpeduncular space at the beginning of the Sylvian fissure and over the pons and the oblongata.

FIG. 211.

Pia from a case of tubercular meningitis. (Obersteiner.)

Lesions in Special Varieties of Leptomeningitis.—In the serous varieties of leptomeningitis the fluid may be turbid, and is probably often of parasitic origin; Quincke believes that the ventricular effusion, which is usually clear, is only exceptionally due to infection. He compares it to the effusion that occurs in angioneurotic edema and the intermittent type of joint affections. As Gowers has put it, it is important to remember that meningeal tubercle and tubercular meningitis are not quite identical, as in general tuberculosis tubercles may be found in the membranes without signs of inflammation, and yet these may cause considerable cerebral disturbance. In tubercular leptomeningitis, while the macroscopic appearances are, as a rule, well marked, the microscope may sometimes be required to decide as to the nature of the lesion. This was true in part of one of the cases reported by Dr. Ott and the writer, and also of Eskridge's case of tubercular cerebrospinal meningitis. At the autopsy in the latter case, tubercles could not be detected by the naked eye by either the reporter or myself, and no exudate was anywhere present; but an expert microscopist, who knew nothing of the case, found tubercles in several of the organs examined, including the brain membranes. In another case, recorded by Galliaux, which was determined microscopically to be tubercular cerebrospinal meningitis, the membranes presented nearly their normal appearance. In tubercular leptomeningitis the most marked appearances are usually present at the base, especially in children. Miliary tubercles may be widely diffused or distributed in many foci. Often the exudate has a jelly-like appearance; at other times it is purulent: great variations are shown, according to the stage of the process. Miliary tubercles are usually minute grayish white nodules, although they sometimes attain a considerable size and may occasionally be so

clustered together as to form a tubercular mass or tumor; and vessels may be obliterated by their pressure, spots of softening being produced in this way. The optic nerve sheaths are often distended. The convolutions may be flattened by intraventricular pressure, or more rarely by subpial fluid or by exudation. The vessel walls are sometimes crowded with leucocytes. Thrombosis even of large arteries occasionally takes place, as in a case recorded by Angel Money, in which a large branch of the right middle cerebral artery was occluded and caused a hemiplegia. This should be remembered when looking for an explanation of some of the cases of complete or nearly complete hemiplegia occurring suddenly in the course of the disease. Even sinus thrombosis may be present, and tubercular deposits may be found on the internal surface of the dura. In nearly all cases tubercular disease is found in other organs, very commonly in the lungs, the spleen, the mesenteric glands, and the intestines. Microscopic examination shows the membranes thickened by an increase of fibrous tissue, infiltration of rounded cells forming pyriform masses around the vessels as they dip down into the cortex; and on the larger vessels of the pia, ovoid masses which do not encroach on the calibre of the tubes. These deposits and the thickened membrane take the stain more readily than do the healthier parts. Goodall has recorded some original observations on the changes that take place in tubercular leptomeningitis. He found in the cortex, just under the meninges, very small round cells which gave off many fine processes, forming a meshwork with neighboring cells. These cells or some of their processes reached the meninges and explained the adherence of the pia to the brain. The processes could not be traced deeper than the third cortical layer. The minute vessels were dilated. In many specimens the nerve cells of the second and third layers were stunted and atrophied, often only the nuclei being left.

Lesions in Epidemic Cerebrospinal Meningitis.—In epidemic cerebrospinal meningitis many of the appearances already described are present. The fulminant malignant cases may not show exudation, but only transudation of coloring matter of the blood, with extreme congestion of the membranes, although numerous microscopical changes may be found. In cases of the average type—those which end fatally in a week or ten days—pus is usually found in the soft membranes both of the brain and of the spinal cord, being most marked in positions already described for other forms. The encephalic dura on its inner surface is sometimes implicated, the spinal dura more commonly, but the pathological changes are generally so limited to the arachnopia that the disease may be considered as chiefly a form of leptomeningitis. The spinal changes may be more decided than those within the cranium; the spinal dura often bulges from the pressure of accumulated fluid. It is not usually adherent

to the pia; an abundant exudate is found in the latter, and this may be very irregularly distributed both vertically and on the various aspects of the cord. The vessels of the pia and of the substance of the cord are deeply injected. The substance of both brain and cord is sometimes softened. Spleen, kidneys, liver, lungs, and other organs may show signs of inflammation or changes such as are found in other severe general diseases. The following summary, from notes made at the time of the autopsy, of the macroscopic appearances in a typical adult case, recorded by Dr. Cahall and the writer, presents a picture of the lesions usually found post mortem. The pericranium was very vascular, and the veins of the dura were gorged with dark venous blood on the outside, while the inner side of the membrane showed marked arterial injection. It was adherent to the convexity of the right hemisphere at the upper border of the quadrate lobule. In front of this lobule, on both sides, were Pacchionian adhesions of the dura to the brain. The frontal lobes, especially on their convexity near the longitudinal fissure, were edematous, with a few slight opacities of the pia. At the base was liquid and semiliquid pus about the optic chiasm, interpeduncular space, pons, and oblongata. All the cranial nerves except the olfactories were bathed in pus, and were more or less softened. The crura, pons, oblongata, and fornix were also superficially softened. The parietal pia (or arachnoid) between the oblongata and the cerebellum was covered by a thick layer of pus. The puncta cruenta were well marked, numerous and dark. The basal ganglia were normal, except for some venous puncta. The fissure of Sylvius presented nothing abnormal on either side, and all the cerebral vessels appeared normal. The spinal cord was flat and spread out on its membranes, and its dural vessels were injected externally and internally. On section of the dura, pus exuded freely. The membranous coverings of the spinal nerves within the canals were almost ecchymotic, most noticeably in those given off opposite the lumbar enlargement, and especially on the left side. The whole cord was surrounded by yellow creamy pus, its lower half being covered dorsally with a yellow gelatiniform pus layer, while in front the same covering was found only over the lower two inches. The spinal vessels were gorged with blood. A few adhesions between the pia and the dura were noticed behind, and many throughout the whole length of the cord in front. The lower ends of both sciatics and of their two divisions (the internal and external popliteal) were removed; they were marked externally by several distended bloodvessels. The second, third, and fourth digital nerves of both feet presented nothing abnormal, but slight pressure at their ends with forceps caused blood to appear.

Microscopical Appearances in Cerebrospinal Meningitis.— The bloodvessels of the membranes and of the substance of the brain

and cord are much dilated. The tissues are swollen, the cells which cause the increase being small with round deeply staining nuclei and a small amount of protoplasm; others, fewer in number, are of epithelioid type. In the spinal tissues a few cells having the appearance of leucocytes with polyform nuclei are found. Single red blood corpuscles free in the tissues, and globular masses which probably represent partly disintegrated red corpuscles, or particles of hemoglobin, are abundant. Cellular infiltration is marked at the roots of the spinal nerves, and especially in the angles between the roots and the cord. The predominating cells resemble lymphocytes or mononuclear cells of granulating tissue. In different positions the cells attain a larger size. Hemorrhages are present both in the spinal and in the cerebral pia. Marked changes in the ganglion cells and axis cylinders are common. The ventral roots show fewer alterations than the dorsal, and not a few of the nerve fibres are swollen. The thickness of the exudate is less on the crests of the convolutions than in the fissures, but it is not uniform anywhere. The vessels of the encephalic pia are much dilated, and the leucocytes with polymorphous nuclei are more abundant in the brain than in the cord. Besides the leucocytes, lymphoid cells and larger cells, some with vesicular nuclei, are found. Fibrin plays a small part in the exudate. (Flexner and Barker.)

Diagnosis.—The diagnosis of acute leptomeningitis is considered under at least three aspects: (1) the differential diagnosis of its various types; (2) the diagnosis from other diseases of the nervous system; and (3) the diagnosis from diseases other than those of the nervous system. Acute purulent leptomeningitis is to be separated from the serous, tubercular, and epidemic forms chiefly by facts relating to the last three. In serous meningitis Quincke advises lumbar puncture and examination of the fluid obtained, to assist in the diagnosis, regarding as suggestive of hydrocephalus from blood stasis a specific gravity of 1009, with an amount of albumen of more than two parts per thousand. Other points in the diagnosis of serous meningitis are given under the clinical description of this disease. Tubercular leptomeningitis is to be distinguished from the simple purulent variety by the history of heredity, by the more frequent prodromes, by the greater tendency of the tubercular disease to attack children, by the more common location of its lesions at the base, by its greater oscillations in temperature, and occasionally by the presence of tubercles in the choroid coat of the eye, as discovered by the ophthalmoscope. Epidemic cerebrospinal meningitis can be separated from other types by the presence of an endemic or an epidemic, by its usually abrupt onset with few prodromes, by the presence of marked spinal symptoms, by the unusual severity of the encephalic manifestations, by the peculiar skin eruptions, and by the absence of a history of the predisposing and exciting

causes of other forms of leptomeningitis. The diagnosis is most difficult in sporadic cases. The epidemic disease, as a rule, runs a more rapid course than the other types. A point of some diagnostic importance is the presence of herpes labialis, which is said to occur in about half the epidemic cases, but is rarely present in the tubercular disease. From culture experiments made from nineteen cases of herpes labialis, Klemperer believes that the eruption represents a peculiar localization of the infecting agent. The diseases of the nervous system from which leptomeningitis must usually be distinguished are brain tumor, abscess, uremia, hydrocephaloid disease, and hysteria ; and among diseases not of the nervous system, typhoid fever, typhus fever, pneumonia, any of the eruptive fevers, and the gastrointestinal diseases of children. The diagnosis of any form of leptomeningitis from other disease should hinge not alone upon the presence of such phenomena as headache, vertigo, and vomiting, delirium and fever, but on such more convincing manifestations as optic neuritis and localized spasms or palsies, either cortical or of the cranial nerves. Optic neuritis may be present in brain tumor, as in leptomeningitis, but it is more common in the former, in which it is usually of a higher grade of intensity. It may be absent in both affections. The pain of brain tumor is more agonizing, and is often more localized by the patient. Tumor cases come on with slower steps, usually having a more or less prolonged prodromal stage. Localizing symptoms are of a more definite and pronounced character. Rapidly growing tumors may be very difficult to diagnosticate from leptomeningitis. Temperature oscillations are much more common in the latter. Abscess of the brain and purulent leptomeningitis may be associated in the same case. When abscess exists as a separate affection, the diagnosis will be based in part upon the fact of its most frequent occurrence in special localities, as the temporal lobe and the cerebellum, where it gives rise to symptoms indicating disturbance of the functions of those portions of the brain. Symptoms indicating implication of the cranial nerves are more likely to be due to leptomeningitis. It must be remembered that encephalic abscess may be latent for a long time. Occasionally uremic attacks simulate fulminant or at least a rapidly developed leptomeningitis. Some of the points of differentiation are for the latter the history, the evidences of disease of the kidneys, heart, and bloodvessels, the less marked fever, the more abrupt convulsive manifestations, the greater tendency to stupor and apoplectiform conditions, and sometimes the presence of albuminuric retinitis. In children suffering from cerebral anemia, as the result of exhausting disease, or from general malnutrition,—a condition first designated by Marshall Hall as *hydrocephaloid disease*,—some of the symptoms suggest tubercular leptomeningitis ; but localizing phenomena are usually absent in the former, and the conditions are

more those of apathy or stupor from profound exhaustion,—fever, headache, retraction of the head, cranial and limb palsies and spasms, etc., being absent or infrequent. The complication of hysteria with leptomeningitis, as with other forms of organic disease, is sometimes observed. Hysteria may simulate tubercular meningitis, although not very closely if a careful study of the patient is made. I have seen a train of hysteroepileptic phenomena, such as pseudoheadache, general spasms with opisthotonos, anesthesias, paresthesias, and paresis, set down as due to meningitis; but such cases are readily cleared up by a careful study of pulse, respiration, and temperature, by a history of the case, and by the absence of the persisting phenomena, local and general, of leptomeningitis. The acute febrile affections are most likely to be taken for leptomeningitis in their early stages, chiefly because of the presence in such cases of headache, delirium, and sometimes of spasms. Prodromes are more likely to be present in meningitis, particularly of the tubercular variety, and of these loss of flesh is especially important. Such symptoms as prolonged irritability and photophobia are more common in meningitis. The temperature, pulse, and respiration are not characteristic, as in some of the infectious fevers, particularly typhoid. In brief, the special features of the general febrile diseases are absent, while those of the different forms of meningitis are present, the mistakes arising from a misinterpretation of a few symptoms. From intestinal diseases the diagnosis is to be made chiefly by the presence of symptoms of local intracranial disease, such as cranial palsies, optic neuritis, and local spastic affections of brief duration.

Prognosis.—The prognosis in all forms of leptomeningitis is comparatively bad, and in tubercular meningitis it is absolutely unfavorable, although Hasse believed in the curability of miliary tubercular meningitis of the pia, and others have reported cases that were cured if they were not instances of mistaken diagnosis. Acute purulent leptomeningitis may sometimes be cured by surgical procedure, and occasionally recoveries are reported from the use of internal and external treatment. Recovery sometimes takes place in serous meningitis either under treatment or by processes which are independent of treatment. The cure may be effected through cessation of the exudation, or by compensation causing widening of the channels of exit. The dilatation of the ventricles may disappear as children grow older. The prognosis of cerebrospinal meningitis is grave, although in epidemics not a few abortive cases are observed, and a fair percentage of cases may recover, particularly towards the decline of the epidemic. The fulminant cases often die within a few hours or days. In all cases the prognosis is serious if stupor and coma come on early. The mortality is greatest in childhood. The tendency to relapse should not be overlooked. One attack does not

give immunity from others, and even when recovery takes place serious sequelæ are often left.

Treatment.—The treatment of leptomeningitis will of course be influenced by its special etiology; thus, when preceded by syphilis, rheumatism, gout, etc., remedies directed to these constitutional states, if they can be used early, are indicated. All forms of acute leptomeningitis present certain common symptoms and indications for treatment. It is first necessary to enjoin complete mental and bodily rest. The patient should be kept in the quietest part of the house; bright lights and all noises and other sources of disturbance should be excluded as far as possible, and sudden alterations in surroundings should be carefully avoided. Food should be administered regularly, but with the least possible disturbance of the patient. Even the mattress should be carefully chosen to make the rest as perfect as possible. Among external applications for the relief of headache and spinal pain are ice bags on the one hand, or very hot applications on the other; one will sometimes afford relief when the other will not. Opium, which in some form is the standby for the relief of pain in meningitis, can be administered by the mouth or hypodermatically. Among the best preparations for administration by the mouth are laudanum, the deodorized tincture, and the salts of morphine or codeine. For hypodermatic use morphine preparations are best. The amount necessary to ease the suffering cannot be gauged by ordinary rules; doses twice or thrice those usually given may be required. Opium is useful as a cerebral stimulant as well as a narcotic in the active forms of leptomeningitis, and especially in cerebrospinal meningitis, in which it sustains the strength of the patient while relieving the pain. It may be given in moderate doses, frequently repeated, with an occasional exhibition of large doses, which, however, may be continuously required. In harmony with others, I have observed the great tolerance of opiates both in tubercular and in cerebrospinal meningitis. When convulsions are severe and recur frequently, large doses of bromides, chloral, or chloralamid should be given, or, if these are already in use, the doses should be increased. Opiates serve sometimes to relieve other symptoms, such as insomnia, restlessness, and delirium, but frequently other drugs are required, and may be given alone or in combination with the opiates. Among these are chloral, cannabis indica, paraldehyde, sulphonal, and trional, which can be given in full doses, using some of the formulas in the section on General Therapeutics. The combination of sulphate of morphine, chloral, and the bromides may prove most efficient; paraldehyde is useful during cerebral excitement; and trional in large dose is an excellent hypnotic, particularly when used in the evening as an addition to the narcotic and sedative treatment which has been pursued during the day. Hyoscine hydrobromate

occasionally proves serviceable, but is not so efficient as it is in mania. For the nausea and vomiting, cracked ice, small quantities of carbonated water, cerium oxalate alone, or with small doses of powdered opium, and bismuth subnitrate or subcarbonate in combination with codeine, and sometimes with salol, may be given. Counterirritants or hot applications over the epigastrium are also serviceable. As the gastrointestinal disturbances are usually of cerebral origin, the remedies which are pushed for the purposes of subduing brain excitability may be of more service than those which are directed especially to the digestive tract. The abstraction of blood, preferably by leeches to the temples or back of the ears, should be resorted to early in sthenic cases of simple or cerebrospinal meningitis. It is contraindicated in the tubercular form, and in all forms in the later stages or when the patient is weak or anemic. In rare instances venesection may be resorted to, as in violent meningitis following sunstroke or traumatism, and occasionally in cerebrospinal cases. For the reduction of temperature the most efficient remedies are sponging, and drugs such as antipyrin, phenacetin, acetanilid, and quinine, the last being used alone or combined with hydrobromic acid. Aconite or veratrum viride may prove useful in sthenic cases. Among remedies which have received almost universal commendation in different forms of meningitis are the mercurial and iodine preparations. They are of especial value in the acute purulent form and in cerebrospinal meningitis, and have caused great apparent improvement even in the tubercular form. The best methods of administering mercury are by inunction and by repeated small doses of calomel, the biniodide or the bichloride. The drug should be pushed until slight constitutional effects are visible. Potassium or sodium iodide or hydriodic acid can be given in doses proportionate to the age of the patient and the severity of the disease. Other valuable measures are the use of iodoform, in doses of two to three grains. until ten or twelve grains are taken in the course of the day ; Lugol's solution, two to three minims, given in simple syrup or in a bland emulsion ; inunction of the shaven scalp with mercurial ointment, ointment of oleate of mercury, or a twenty per cent. iodoform ointment. A good combination in almost any form of leptomeningitis is that of the iodides and bromides with narcotics, such as cannabis indica, hyoscine, or morphine. Mercury and opium are frequently given together advantageously. Surgical treatment, while not applicable to epidemic cerebrospinal meningitis, may be demanded in simple purulent leptomeningitis, and offers some hope even in the tuberculous form of the disease. If an abscess can be localized, or if the purulent leptomeningitis is believed to be confined to an accessible area, trephining can be resorted to for the removal of the pus, or for antiseptic irrigation. Lanphear,

Keen, and Senn have advocated the surgical treatment of tubercular leptomeningitis, recommending opening the skull and washing out the meningeal spaces with iodoform and glycerin emulsion or with other germicidal preparations. Of course the surgical treatment of aural, nasal, or any other form of disease which has led to the meningitis must be carefully considered. Sometimes a leptomeningitis will be present on the side of the brain opposite to that on which the aural or mastoid disease is located, as in a case seen by me in consultation, in which purulent disease of the left ear was present, and among other severe symptoms were paralysis of the limbs and unilateral convulsions on the same side as the aural disease. Trephining of the mastoid did not afford relief. The patient died, and a purulent leptomeningitis was found extending from a point an inch and a half back of the tip of the left hemisphere to the parieto-occipital fissure, and from the longitudinal fissure nearly to the Sylvian. This case might have been successfully treated by trephining over the area of leptomeningitis, which was large but was strictly limited to a region which could readily have been reached and irrigated. Ord and Waterhouse successfully treated tuberculous meningitis by trephining and irrigating the subarachnoid space. The first case of leptomeningitis thus treated was by Barker. The treatment of serous meningitis consists, for the acute forms, in antiphlogistic measures, the application of cold and of leeches, complete mental and bodily rest, and the use of intestinal derivatives. Mercury by inunction or hypodermatically, pilocarpin, quinine, and antipyrin during febrile exacerbations, flying vesicants, iodine, and cautious inunctions with tartar emetic ointment, are included in Quincke's recommendations. In cases of imminent danger he recommends lumbar puncture, removing from five to eight cc. of fluid through a hollow needle introduced at a distance of one cm. from the middle line beneath the second, third, or fourth lumbar vertebra. For the prevention of tuberculous meningitis all measures which tend to improve constitutional conditions, such as good food, fresh air, and good ventilation, are important. It is possible that great good might be done in aborting acute leptomeningitis, or in reducing its gravity, by treating it early from a bacteriological point of view. In the presence of an epidemic and in the early stages of infectious forms of meningitis, the administration of mercury, potassium bicarbonate, sodium salicylate, and similar drugs might in this way do something.

CHRONIC LEPTOMENINGITIS.

General Considerations.—Hemorrhagic internal pachymeningitis is usually a chronic affection, as is also external leptomeningitis, or arachnitis so called, the latter often being the result of recurring attacks of acute inflammation or congestion. Besides these

diseases, other forms of membranous inflammation to which the term chronic properly belongs are met with somewhat frequently, particularly in the victims of alcoholism, traumatism, and insolation. If infection is the almost invariable cause of meningitis, either acute or chronic, the above and other etiological influences may so diminish cerebral and systemic resistance as to allow the omnipresent germs of infection to originate and maintain chronic inflammation of either encephalic or spinal membranes. The form of chronic meningitis to which attention is here briefly directed is that which affects the entire pia, but it is rarely confined to that membrane, the brain substance often and the dura less frequently taking part. Chronic leptomeningitis may be either *primary* or *secondary*, but it is commonly the latter. For practical reasons, nervous syphilis, alcoholism, and cerebral traumatism will be considered in special sections, and because of this the subject of chronic meningitis can be dismissed here more briefly. It is discussed in this connection in pursuance of the plan of making localization and diseases of special structures the chief method of classification. Chronic leptomeningitis of a low grade is a common pathological lesion in a large number of those who die insane; although even in them it is not to be regarded always, or even often, as the special pathological cause of the mental disorder, but rather as a condition which has developed step by step with the progress of the mental disease—a part of the general structural breakdown of the patient. The changes in the membranes in these cases are brought about not infrequently by the recurring congestions which accompany the waves of excitement through which the patients pass. According to Bevan Lewis, the pia is abnormally thickened in fully forty-eight per cent. of those dying insane, partly from fibrinous exudates which have organized, partly from plastic lymph, and often from an edematous swollen condition of the conjoined soft membranes. These statistics include many general paralytics, but also many cases of acute mania or acute melancholia. (Batty Tuke.)

Clinical History.—Because of the variations in the intensity of the inflammation and the irregularities as to location and diffusion of the lesions in different cases, it is almost impossible to present the symptomatology of chronic meningitis in a thoroughly satisfactory manner. The local symptoms will be those presented by different types of acute leptomeningitis; the general symptoms are usually much less severe; and both local and general manifestations are modified by the mental and physical conditions which are slowly induced. Like the acute form, it may be of the convexity or of the base, or it may be focal in any position. Syphilitic leptomeningitis, when focal, often shows an inclination towards certain localities, as the foramen magnum, the junction of the pons and the oblongata, or of the pons and the crura, and the prefrontal, the Rolandic, or

the Sylvian convexity. The symptoms in any type may vary according as one or other of these positions is attacked. When the prefrontal region is the seat of the disease, they will be largely psychical, such as apathy, inattention, and mental slowness; when the inflammation is in the motor region, various forms of paresis and spasm may be present or come and go; or the manifestations may be visual, auditory, aphasic, gustatory, or olfactory, when other cortical areas are invaded. If basilar, the cranial nerves will suffer most, and alternate hemiplegias of various types, and usually imperfectly developed, may be present, as oculomotor, motor trigeminal, facial, or abducens paralysis of one side, with hemiparesis of the limbs of the other. The form of basilar meningitis described by Gee and Barlow as occurring near the foramen magnum, to which reference has already been made, may be a chronic affection, and is sometimes spoken of as chronic infantile meningitis. Certain symptoms would seem to be common in almost all types of chronic leptomeningitis. These are dull headache, mental changes, muscular weakness, even without true paresis, vertigo, more rarely nausea or vomiting, and tinnitus. Optic neuritis of a low grade is a common but not a constant sign. Occasionally a case of chronic leptomeningitis may present symptoms of striking or unusual character, as in a case reported by me of athetoid spasm, myotonia, and diffuse bilateral disturbances of sensation, which was found to be due to chronic convexity meningitis of both hemispheres with cortical and subcortical softening, the lesion being most marked in the posteroparietal region. This patient nine years before his death had a sunstroke followed by a fit; subsequently he had similar seizures, at first with great

FIG. 212.

Myotonia and athetoid spasm in a case of convexity meningitis, with cortical and subcortical softening.

frequency and later at longer intervals. He gradually developed a spastic state and athetoid movements, with paresis first in one limb and then in another, and eventually in the face, chiefly on the left side. Violent choreoid and athetoid movements were produced by willed efforts, and by manipulation, and sometimes were apparently spontaneous. When he opened his mouth the muscles of the face, neck, tongue, and extremities took part in a series of athetoid and chronic contractions, much more marked on the left side of the body, although distinctly present on the right. (Fig. 212.) Knee jerk and muscle jerk were increased; ankle clonus and front tap were present. Percussion of the muscles gave phenomena similar to Erb's myotonic reactions. At intervals he had attacks of Jacksonian epilepsy, and shortly before his death had general convulsions. Strange to say, he did not suffer from chronic headache. In this case, in addition to a true leptomeningitis, shown by deep injection of the pia, and thickening and infiltration of the membrane with plastic lymph, which was more or less adherent to the brain substance, cortical and subcortical softening were present. The symptoms were therefore both meningeal and subcortical, but the case was primarily one of a severe form of verticalar meningitis. As chronic leptomeningitis is frequently secondary to other affections, such as brain tumor, atrophy, and fracture, the symptoms will be complicated with those of the primary affection. Grave hysterical manifestations may be present and for a time may overshadow the organic affection. Under a temporary exciting cause, or from a sudden increase in the virulence of the infecting agents, the sufferers from chronic meningitis may have acute attacks attended with headaches, convulsions, increase of optic neuritis, fever with delirium, or temporary stuporous states. The symptoms of these paroxysms are much like those which occur in the course of brain tumors.

Etiology.—Chronic leptomeningitis, like acute leptomeningitis, is usually due to infection; but various causes already referred to, such as traumatism, alcoholism, and insolation, play an important part in the development of the disease. Insolation is a more frequent factor in the production of pachymeningitis. Chronic leptomeningitis, like acute leptomeningitis, has been ascribed to gout, rheumatism, diabetes, influenza, typhoid fever, scarlet fever, etc. It is usually a disease of adults. It occasionally follows in the wake of acute leptomeningitis.

Pathological Anatomy.—The pathological appearances, which vary according as the affection is tubercular, serous, or purulent, differ from those of the acute form already described chiefly in being less marked in character. In syphilitic cases fibrocaseous and gummatous thickenings are common over the surfaces of the convolutions, and infiltration of the cortex is not infrequently present. Dura and

pia are often agglutinated, and the latter membrane may be continuously or irregularly adherent to the cortex. In advanced cases many of the vessels which constitute the pial network are obliterated, cortical and subcortical softening resulting. Cranial nerves and vessels may be bound down by old or recent exudate. Recent infiltrations of the pia may be present in some cases. Peculiar looking cells are often found aggregated into spherical clusters, which become homogeneous and finally calcified. They are often surrounded by very small cells and recently formed fibrous tissue. (Ziegler.) Effusions of serum, pus, or lymph may be present, but the latter is most constant in chronic cases. Pacchionian granulations are frequently increased and enlarged. The lesions may be found in any location, and may be variously diffused. In many cases of chronic alcoholism, and in some cases of insanity to which reference has been made, the lesions are not of the marked character which is indicated by the above description, but consist chiefly of opacities of the parietal layer of the pia, with some thickening and deposits in the visceral portion of this membrane. Vessels as well as membranes are sometimes changed by chronic inflammatory processes, some forms of chronic leptomeningitis being part of a general process which attacks vessels and viscera or any of the bodily organs or tissues. In the syphilitic nodose periarteritis which has been best described by Ziegler and Bruce, marked changes may be found in the membranes as well as in the vessels. In chronic leptomeningitis of long duration, atrophy of the brain is commonly present. Convolutions, lobes, or even the entire brain may present a shrunken appearance, and the absent cerebral substance is substituted by cerebrospinal fluid.

Diagnosis.—The diagnosis of chronic leptomeningitis may be difficult. It must be based chiefly on a study of causation; on the history of a slow development; on the age and sex of the patient; on the persistence of certain symptoms, such as mental change, dull headache, or discomfort in the head; on the presence of cortical or cranial nerve symptoms, and of low grade optic neuritis. In the early stages of general paralysis of the insane, the patient may be supposed to be suffering from a form of chronic curable leptomeningitis, but the progressive development of the physical and mental symptoms of this form of insanity will usually soon clear up the diagnosis. The diagnosis will need to be made mainly from such affections as brain tumor, chronic Bright's disease, and hysteria or neurasthenia. From tumor of the brain it is chiefly to be distinguished by the greater intensity and the more focal character of the symptoms in tumor cases. Optic neuritis is usually of higher grade in brain tumor. The symptoms of the two affections may be commingled. Occasionally localized syphilitic meningitis cannot be distinguished from syphilitic brain tumor. The headache, mental

changes, eye changes, and convulsions which may occur in the course of chronic nephritis have led to its being confused with chronic leptomeningitis; but the two affections can usually be differentiated by close attention to the signs and symptoms referable to the disease of the kidneys, as by investigation for albumen and casts, by examination of the eyes for albuminuric retinitis, and by the effects of medication. Occasionally chronic leptomeningitis is set down as hysteria or neurasthenia, but a close study of the patient will enable the practitioner to determine the presence of organic disease. In some cases of neurasthenia, particularly if the patient is suffering from abnormal refraction, the eye grounds present an appearance somewhat like that of a true optic neuritis of low grade. In children and young adults the differential diagnosis of tubercular leptomeningitis from a form of hereditary leptomeningitis, due to inherited syphilis, may be of great importance. Probably in most cases an absolute diagnosis cannot be made except by the therapeutic test, but the usual signs of inherited syphilis, such as nasal flattening and notched teeth, will be helpful. In a doubtful case it is best to treat the patient with large doses of mercury or of the iodides, especially the latter. It is not improbable that many of the cases of tubercular meningitis reported as cured have been of this character; but that they do not all belong to this category is shown by the case reported by Freyhan, who made a diagnostic puncture in the lumbar region and obtained a fluid in which were found pus corpuscles and tubercle bacilli. This patient is reported to have recovered under treatment.

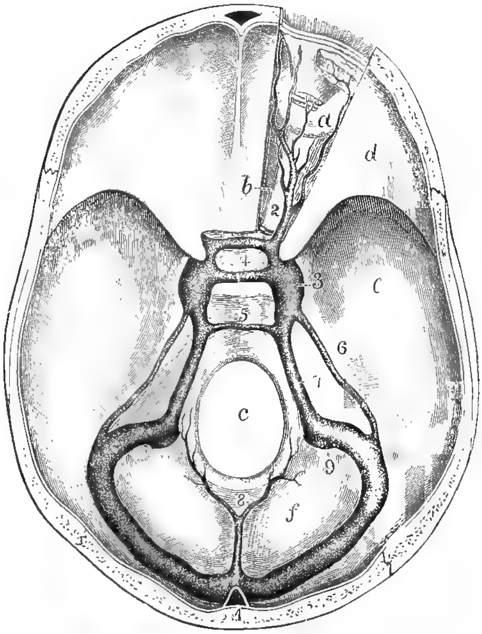
Prognosis.—Little need be said about prognosis, which is usually more or less unfavorable. Active treatment in syphilitic cases and in those due to insolation or traumatism will sometimes effect a cure or a proximate cure, and will at least make life much more bearable.

Treatment.—Mercury and the iodides, especially when syphilis is suspected, but in any case for their absorbent powers, may be used either cautiously or boldly according to indications or the general strength of the patient. Although seldom curative, they often do good by preventing exacerbations and decreasing the intensity of the symptoms which cause suffering. Ergot and the bromides may be used with good results at intervals. Persistent counterirritation, particularly with the Paquelin cautery at the back of the neck, will be found to give great relief. Hot or warm baths, a sojourn at hot mineral springs, a change of climate in cases which are much affected by season and weather, are all worthy of trial. Much of the treatment will necessarily be symptomatic, as, for instance, for the relief of pain and insomnia, and may involve a careful use of opium, chloral, trional, and other hypnotics, sedatives, and narcotics. As the general health of patients is always much impaired, nutrients and tonics will often prove of value.

THROMBOSIS OF THE INTRACRANIAL SINUSES.

The Dural Sinuses.—As the intracranial sinuses are constituted by folds of the dura, it is convenient to consider the diseases of these large bloodways in connection with affections of the brain membranes. The most important sinuses both medically and surgically are about fifteen, although the number is variously given by different anatomists. Allen makes it sixteen. The variations are caused by some considering as separate sinuses parts which others include under one. Basal and vertical views of the most important sinuses are shown in Figs. 213 and 214. The feeders of these sinuses are the veins of the brain, skull, orbit, ear, and other parts external to the skull. The sinuses differ from veins in not accompanying arteries, in not having valves, and in not having muscular coats. They are lined, like the veins, with a delicate membrane which may become the seat of inflammatory disease. It will not be necessary fully to describe these sinuses; the illustrations and legends* will be sufficient to call to mind the most important anatomical points. The term *sigmoid sinus* is applied to the S-shaped portion of the lateral sinus which lies in the sigmoid groove of the temporal and occipital bones, and is in direct relations with the mastoid and jugular regions. This curved portion of the sinus is not well shown in Figs. 213 and 214, but is seen in Fig. 215, which is a diagram slightly modified from part of an excellent scheme by

Fig. 213.

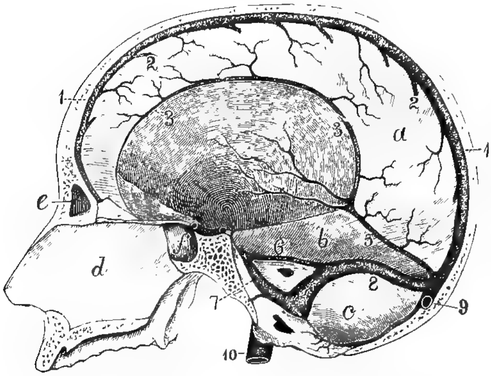


Basal cranial sinuses, showing also the right orbit: 1, torcular (torcular Herophili or confluence of the sinuses); 2, ophthalmodural (ophthalmic) vein; 3, cavernous sinus; 4, circular sinus; 5, preoccipital (anterior occipital) sinus; 6, 7, superpetrous and subpetrous (superior and inferior petrosal) sinuses; 8, postoccipital (posterior occipital) sinus; 9, commencement of internal jugular vein; *a*, eyeball; *b*, optic nerve; *c*, occipital foramen; *d*, *e*, *f*, anterior, middle, and posterior fossæ. (Leidy.)

* Browning has extended the simplified terms of Wilder, usually mononyms, to the veins, sinuses, and meningeal vessels. For meningeal he has substituted dural, as the sinuses and so-called meningeal vessels lie wholly in the dura. The simplified terms are given first in the legends and text, the commonly used names in parenthesis.

Macewen of the intracranial and extracranial venous anastomoses. It is of the greatest importance to understand the position and relations of this sinus, lying as it does so close to the middle ear, the mastoid antrum and cells, all of which are so frequently the seats of infective inflammation. A thrombus occasionally extends from the superior petrosal into this sinus, while the reverse process is of frequent occurrence. One lateral sinus, generally the right, is often larger than the other.

Fig. 214.

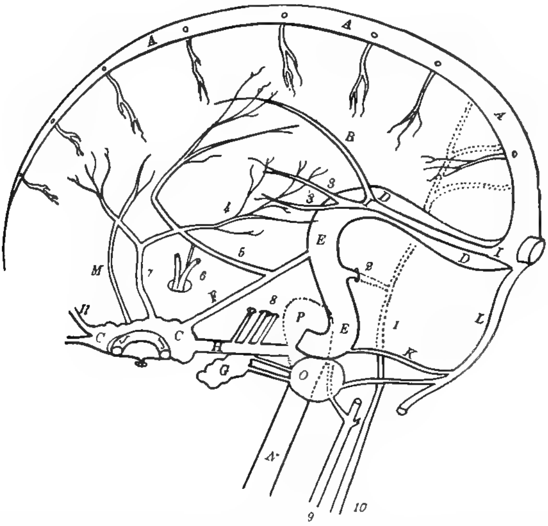


Vertical view of the dural sinuses: 1, longitudinal (superior longitudinal) sinus; 2, termination of supercerebral (superior cerebral) veins; 3, falcial (inferior longitudinal) sinus; 5, tentorial (straight) sinus; 6, 7, superpetrous and subpetrous (superior and inferior petrosal) sinuses of the right side; 8, right lateral sinus; 9, commencement of the left lateral sinus; 10, internal jugular vein; *a*, falx; *b*, tentorium; *c*, cerebellar fossa; *d*, partition of the nose; *e*, frontal sinus. (Leidy.)

The Interrelations of the Veins and Sinuses.—It is important to understand the relations of the veins of the brain, of the skull, and of the parts outside of the skull, to the sinuses. According to Duret, all the veins of the cerebral gyres in their confluences are disposed after two plans, perpendicular to one another, one vertical and mesal, which is connected with the longitudinal sinus and veins of Galen, the other posterior and horizontal, situated at the base of the brain and connected with the cavernous and petrous sinuses. The veins of the convexity and mesal surface in general empty into the longitudinal sinus; those of the inferior surface of the cerebrum—of the frontal, temporal, and occipital lobes—into the lateral, petrosal, and cavernous sinuses. Communication is free between the superior and inferior cerebral veins. The veins of the ganglia and interior of the brain unite to form the veins of Galen and pass out of the great transverse fissure to enter the tentorial or straight sinus, commonly, however, uniting to form one vein before entering the sinus. Supercerebellar (superior cerebellar) veins terminate in the tentorial or straight sinus; the subcerebellar (inferior cerebellar) veins in the lateral sinuses; the paracerebellar (lateral anterior cerebellar) veins in the superpetrous (superior petrosal) sinus. Duret found that the cerebral veins had more important anastomoses than the arteries, but that individual variations were numerous. Often it is thought that two branches anastomose when they are simply side by side at the bottom of a fissure. The veins of the hemisphere send off arborizations in the convolutions. The ophthalmodural or ophthalmic

veins empty into the cavernous sinuses, as do also the veins of the external ear by way of the petrous portion of the temporal bone. The veins of the mastoid regions empty into the lateral sinuses. The cephalic extremity of the longitudinal sinus communicates by the foramen cecum with the nasal fossæ. The veins of the diploë communicate with many of the sinuses. An important fact to bear in mind in connection with cerebral thrombosis is that the veins empty into the sinuses in a direction opposite to that of the blood current in the latter, also that the most important sinuses are crossed

FIG. 215.



Scheme showing some of the most important relations of the intracranial sinuses and veins: *A, A, A*, longitudinal (superior longitudinal) sinus; *B*, falcial (inferior longitudinal) sinus; *C, C*, cavernous sinus; *D, D*, lateral sinus (straight portion); *E, E*, sigmoid sinus (curved portion of lateral sinus); *F*, superpetrous (superior petrosal) sinus; *G*, basilar sinus; *H*, subpetrous (inferior petrosal) sinus; *I, I*, tentorial (straight) sinus; *J, J*, circular sinus; *K*, marginal sinus; *L*, occipital sinus; *M*, sphenoidal (sphenoparietal) sinus; *N*, internal jugular vein; *O*, foramen magnum; *P*, jugular bulb; 1, occipital vein; 2, mastoid vein; 3, 3, veins of Galen; 4, temporosphenoidal veins; 5, anastomotic vein; 6, medidural (middle meningeal) veins; 7, medicerebral (middle cerebral) vein; 8, veins from the labyrinth; 9, vertebral vein; 10, posterior external jugular vein; 11, ophthalmodural (superior ophthalmic) vein. The frontal, parietal, and occipital superior cerebral veins are shown communicating with the longitudinal sinus. (After Macewen.)

by white fibrous bands or trabeculæ. Roy and Sherrington found that closure of both external jugulars caused expansion of the brain. It is therefore important to remember that pressure in the veins influences the volume of the brain. All the blood in the intracranial sinuses eventually finds its way into the jugular veins.

Definitions and Varieties.—Sinus thrombosis, or clotting of the blood in the cerebral sinuses, is always a serious and often a fatal disease, and in its different forms is of both medical and surgical interest. It may be *primary* or *secondary*. The primary form

is often spoken of as *marantic* or *marasmic*, and the secondary as *infective* or *inflammatory*.

Clinical History of Primary or Marantic Thrombosis.—A patient attacked with this disease generally becomes somnolent and later stuporous and comatose, and may have as other symptoms convulsions, paralysis, retraction of the head, edema of the scalp, and bleeding from the nose. In the majority of cases fever is not present or is scarcely discernible; occasionally the temperature is raised one or two degrees. The disease may be present with scarcely any symptoms except stupor deepening into coma, when it is extremely difficult to diagnosticate it from anemia of the brain. In adults paralysis is comparatively common. The duration is from three or four days to several weeks. Occasionally young children recover, sometimes having as sequels impairment of mental powers and a tendency to convulsions. Both spasm and paralysis are probably often due to the meningeal hemorrhages which occur as a result of the thrombosis. Besides the usual coagulability of the blood, the heart may be weakened, and the total amount of blood circulating in the body may be reduced. Primary thrombosis is of most frequent occurrence in the longitudinal sinus, but in rare instances it has been observed in the lateral and cavernous sinuses.

Clinical History of Secondary Sinus Thrombosis.—Secondary sinus thrombosis has symptoms which are necessarily commingled with those of the diseases from which it originates or with which it is associated, as affections of the external and internal ear, cranial caries, cerebral or cerebellar abscess, carbuncle, facial erysipelas, or other local affections of the head or face associated with suppuration. Headache, muttering delirium, stupor, and coma are general symptoms in many of the types of infective thrombosis, and are sometimes due as much to an accompanying meningitis as to the thrombosis. Occasionally the patient is maniacal, and symptoms of general septicemia, such as chills and irregular fever, may be present. Three types of secondary or infective thrombosis are described by Macewen,—the *pulmonary*, the *abdominal*, and the *meningeal*. In the pulmonary type the symptoms, which, in the first place, are due to the plugging of the small vessels and the formation of infarcts in the lungs, are irritative cough, scattered stitches of pain or diffused pain, and in a short time prune juice expectoration, which soon becomes of a brownish gray hue, owing to the admixture of pus, and which is found to be swarming with microorganisms. A purulent generalized pneumonia may result. Great fetor of the breath and diarrhea with fetor of the discharges from the bowels are usually present. Acute pain in the region of the liver may be complained of, and is due either to the extension of inflammation from the lungs or to primary congestion or inflammation of the liver, but hepatic abscess is rare. The brain usually remains clear until the end of the

disease. The abdominal or typhoid type shows characteristic symptoms at the end of the first or at the beginning of the second week. They are such as dry furred tongue, anorexia, vomiting, abdominal pains, diarrhea,—the stools being very fetid in character,—muttering delirium, languor, stupor, and a measly rash. In brief, the symptoms are so much like those of typhoid or enteric fever that a mistaken diagnosis has not infrequently been made. The headache is of violent character, vomiting is frequent, the temperature is high, but rigors are uncommon. In the meningeal type excitement and irritability, a mixture or alternation of spasm and paresis, great hyperesthesia, and disturbances of the cranial nerves are prominent. Sometimes spinal leptomeningitis may accompany the intracranial affection, when spinal symptoms will be present. The patient becomes comatose towards the end, and throughout the disease psychical and other cerebral symptoms are more common than in either the pulmonary or the abdominal type. The symptoms of the different types may be commingled in any case, and will of course vary with the particular sinus affected, as will be next considered.

Longitudinal Sinus Thrombosis.—Certain local symptoms will depend on the special sinus affected; some of the sinuses even present differences in the general symptoms. Owing to its situation and great size, for example, the longitudinal gives positive disturbances of the character already indicated in considering marantic thrombosis. From a study of the symptoms observed in three cases with autopsies, Du Pasquier believes that somnolence, coma, a state of rigidity, a fixed and prolonged attitude in extension or flexion, champing of the jaws, blepharospasm, and tremor of the fingers indicate the existence of a clot in the longitudinal sinus. Among local manifestations attributable to the occlusion of this sinus are epistaxis, edema in the region of the nose and temple, and in young children prominence of the fontanelles.

Cavernous Sinus Thrombosis.—Lying mesad of the cavernous sinus is the cavernous plexus of the gangliated system of nerves, and the abducens nerve; while on or in its lateral walls run the oculomotor, the pathetic, and the first or ophthalmic branch of the fifth nerve, and therefore interference with these nerves may give very definite localizing phenomena. One or both eyes may be immovable; one may be more prominent than the other; ptosis may appear on one side and later on both. Optic neuritis may be shown by one or both eyes. Supraorbital or infraorbital pain may be marked, especially at the onset; and later, anesthesia of the conjunctiva and cutaneous distribution of the first division of the fifth nerve may occur. Other most important symptoms are dependent upon the communications of the sinus with the ophthalmic vein, which supplies parts both within and without the orbit. Exophthalmus is sometimes so marked as to suggest an orbital growth.

The eyelids and parts about the orbit may be greatly swollen. Thrombosis of the nasal, angular, or temporal veins, with or without suppuration, may be present. The frequency with which symptoms at first unilateral become bilateral is an important point in the study of cavernous thrombosis. Thrombosis of the petrosal sinus may be associated with or may extend into the cavernous sinus or from the latter into the former, so that in petrosal thrombosis some of the symptoms of cavernous thrombosis may be present.

Lateral Sinus Thrombosis.—Thrombosis of the lateral sinus is usually of its sigmoid portion, and hence is often spoken of as *sigmoid sinus thrombosis.* It is commonly associated with diseases of the mastoid or of the middle ear, and will in the first place have symptoms indicative of these affections. The region of the mastoid may be swollen and edematous, and not infrequently the headache or head pains will be more localized to the posterior portion of the head. In some cases where the thrombosis extends downward into the internal and common jugular veins, the latter filled with thrombic masses may be palpated; in others sensitiveness to pressure in the retromaxillary fossa and on the inner border of the sternocleidomastoid muscle, and edema of the soft parts of the neck, may exist. Sometimes hoarseness and dysphagia are observed. A benign thrombus filling the transverse occipital sinus cannot be diagnosticated, as a rule. If it extends through the emissary veins of this sinus and their anastomoses, it may form induration in the connective tissue at the back of the neck resembling a phlegmasia alba dolens. (Rissler.) It is important to remember that edema and pain in the mastoid may be absent in lateral sinus thrombosis, and that these symptoms may be more prominent in suppuration and inflammation of the mastoid cells. The edema in thrombosis is often slight. In chronic otitis media the cessation of otorrhea coincident with accession of persistent otalgia extending into cephalalgia, high temperature with marked fluctuations, vomiting, and rigors, ought to be regarded as pointing in the direction of thrombosis of the sigmoid sinus. (Macewen.)

Etiology.—Primary thrombosis is often called marantic or marasmic, because of its frequent occurrence as the result of exhaustion or malnutrition, and is commonly associated with infantile diarrhea, wasting diseases such as cancer, phthisis, and pernicious anemia, or more rarely with the acute infectious diseases. It is usually a disease of either early infancy or extreme old age, but may occur at any period of life. Infective or secondary thrombosis may arise by extension of inflammation from the neighboring parts to the walls of the sinus, which become inflamed, the blood coagulating and adhering to the inner coat of the vessel. Thrombosis arising in this way is frequently called phlebitic. A second method of origin is by the formation of a venous thrombus which soon extends into

the sinus. Pressure on a vein or sinus is an alleged cause of thrombosis, but it certainly rarely originates in this way. Diseases of the ear have a most important practical bearing upon secondary sinus thrombosis as well as upon meningitis. All statistics tend to show the preponderating rôle played in these diseases by affections of the middle ear including the tympanic cavity and its offshoots. Out of 43,730 aural cases analyzed by Bürkner, sixty-six and nine-tenths per cent. were of middle ear disease. Thrombosis and intracranial meningitis rarely follow acute primary suppurative disease of the ear. They are most common in the course of chronic aural affections, for the obvious reason that, lining membrane and bone being more or less destroyed, the lymphatics and other channels for the removal of infectious material are no longer sufficient for this purpose, and contamination by way of the bloodvessels which course through the bones and by way of the labyrinth and nerve sheaths readily takes place. The lymphatics as well as the veins and the arteries play their part in propagating inflammation from the tympanum to the brain and meninges. (Barker.)

Pathology.—In primary as in secondary thrombosis it is probable that the initial state is a radical alteration of the endothelium. (Ball.) The affected sinus, commonly the longitudinal, is found filled with a mass of blood in various stages of change. In recent cases of marantic thrombosis the clot is usually soft and dark, but as time progresses it becomes yellowish and friable. Sometimes it is large, filling the sinus or even extending beyond it into other sinuses and veins, but it varies much in this respect. Appearances of inflammation are absent in marantic cases. Edema, hyperemia, and cortical capillary hemorrhages occur as the result of the obstruction. When recovery takes place the brain may become atrophied and hardened over irregular areas. As the result of infective thrombosis, great changes take place in the walls of the sinus, which may disintegrate to such an extent as to cause a break in its continuity, although hemorrhage is uncommon as the result of this, because of the coincident coagulation of the blood. The clot within the sinus soon disintegrates and becomes purulent. It swarms with microorganisms, which may infect the system in at least three ways: (1) by way of the tributary veins, (2) by portions of the disintegrated clot being carried into the general circulation, and (3) by spreading of the infective matter through the walls of the sinus into the neighboring parts. Variability in the character and virulence of thrombosis and other intracranial affections may be dependent in part on differences in the nature and activity of the microorganism present. Barker and Rohrer hold to the saprophytic nature of the bacilli usually found in aural discharges.

Diagnosis.—In children marantic thrombosis most resembles the hydrocephaloid disease of Marshall Hall, or profound anemia of the

brain, the two being often associated. The persistence of serious symptoms such as coma, spasm, and paralysis points to thrombosis. The most important diagnostic symptoms are those which distinctly indicate interference with the circulation, as marked fulness of the nasal, frontal, or temporal veins. In adults the disease is confounded with arterial thrombosis, from which it is to be differentiated by the less frequent occurrence in the latter of stupor, coma, and spasm, and by the more definite character of the paralysis in the former, which can usually be referred to some well known arterial distribution. Sinus thrombosis also may occur in adults without the arteriocapillary sclerosis which is so common in the arterial disease. The occurrence of convulsions towards the end rather than at the beginning of an acute infantile affection is an important diagnostic point in favor of primary thrombosis. The diagnosis of secondary thrombosis will be made from the history of the case, from the general symptoms which are present, and from the symptoms which indicate the involvement of special sinuses. With regard to the history of the case, the presence of disease of the middle ear or of the mastoid, of facial erysipelas, of disease of the orbit or of the mouth, or of any purulent affection of the head, would turn the attention of the physician to the probable presence either of thrombosis or of meningitis, or of both. The general symptoms of sinus thrombosis when secondary are in the main those of meningitis, such as headache, delirium, sometimes stupor and coma, and irregular fever. If purulent localized meningitis is present, the symptoms may indicate the parts affected, as unilateral paralysis, or paralysis of the cranial nerves, or affections of various cortical areas, as of the auditory or visual centres. In the diagnosis of special varieties of sinus thrombosis close attention must be also paid to the localizing symptoms. Thrombosis of the cavernous sinus might possibly be confused with several other affections, as local orbital disease, focal meningitis, neuritis of the third, fourth, or sixth nerves, tumor, aneurism, or even nuclear ophthalmoplegia. Careful examination will usually be sufficient to exclude a gross orbital lesion. Focal meningitis of the region of the cavernous sinus may be very difficult to differentiate, and is often associated with occlusion of the sinus. In meningitis the symptoms which indicate involvement of the nerves will be likely to precede those which show the presence of obstruction, such as swelling with edema. Optic neuritis is more likely to be present with meningitis than with pure thrombosis, but may be a symptom in either. Neuritis, even when syphilitic in origin, rarely attacks more than one cranial nerve at the same time. The limitation of the phenomena to those parts within and around the cavernous sinus is, on the whole, against the diagnosis of tumor, although of course a growth may have any localization within the cranial cavity, making the diagnosis

in some instances extremely difficult. Optic neuritis is of more frequent occurrence in tumor. The absence of bruit and tinnitus will assist in excluding aneurism. Nuclear ophthalmoplegia will be excluded by the fact that it runs a chronic progressive course, usually becoming bilateral, and by the absence of symptoms of irritation and obstruction. In considering the diagnosis of thrombosis of the lateral sinus, the most important points are the history of purulent discharge from the ear; the sudden onset of the illness with rigor, vomiting, and pain in the affected ear, oscillatory temperature, local edema and tenderness over the mastoid and the internal jugular; tenderness on deep pressure at the posterior portion of the mastoid, and below the external occipital protuberance; stiffness of the muscles of the back and side of the neck. (Ballance.) Puncture with a hypodermatic needle will determine whether the venous channel is occluded. Symptoms attendant upon emboli being carried to other parts of the body will be of value for the diagnosis of thrombosis of the lateral or of any of the larger sinuses.

Differential Diagnosis of Primary and Secondary Thrombosis.—The following table from Macewen shows at a glance the main features which separate primary or marasmic from secondary or infective thrombosis:

Marasmic (Primary).	Infective (Secondary).
1. Chiefly affects the azygos* sinuses.	1. Chiefly affects the dual sinuses.
2. The clots tend to organization or are absorbed.	2. The clots tend to purulent disintegration.
3. Hemorrhages into cerebral cortex in about half the cases.	3. Hemorrhages into brain seldom occur.
4. Tendency to produce brain softening.	4. No tendency to brain softening.
5. Seldom purulent infection as a sequence.	5. Purulent infection common; septic or infective emboli.
6. No accompanying leptomeningitis, cerebral or cerebellar abscess.	6. Often coincident purulent leptomeningitis, cerebral or cerebellar abscess.

Prognosis.—The prognosis of both primary and secondary thrombosis is bad, but recoveries from either may occur. The recovery from marantic thrombosis is usually not complete, the patient being left with some form of athetoid, choreoid, spastic, or paralytic disease. It gives rise to some of the forms of infantile spastic paralytic disorders. Recovery from secondary thrombosis is usually due to successful surgical interference.

Treatment.—If either primary or secondary thrombosis is discovered or suspected during the formative stage, something may be done for its relief. Stimulants, concentrated liquid nourishment, and constant application of heat to the head are useful measures.

* The term *azygos* is applied to parts that are single,—that is, not in pairs.

Among the most efficient remedies to improve and stimulate circulation and aid general nutrition are drugs such as strychnine, strophanthus, digitalis, arsenic, and iron. Gowers suggests that attention should be paid to posture so as to aid the flow of blood in the sinus affected; thus, flexion of the neck should be avoided when the thrombosis is of the longitudinal sinus. It is no longer true, as was asserted by Huguenin nearly twenty years ago, that we can do nothing to overcome the vascular occlusion itself, supposing it to have been established. Thrombosis of the lateral (sigmoid) sinus has been treated successfully by operation by Barker, Ballance, Salzer, Clutton, Macewen, and others, although in many cases operation has failed. Usually the jugular vein is tied, and the corresponding lateral sinus exposed and washed out through the jugular above the ligature. When the thrombotic clot has extended into the petrosal and other sinuses, or has caused purulent meningitis and abscess, the chances of success are poor; although it is possible to operate successfully in the same case for both thrombosis and abscess or purulent meningitis. The prophylaxis of infective sinus thrombosis, as of purulent leptomeningitis and of intracranial abscess, is of first importance, and may demand the attention of both the general practitioner and the surgeon. Whenever possible, the formation of primary infective foci should be prevented; but if these should be present, prompt and radical measures should be taken for their removal. To carry out these indications involves the most careful antiseptic treatment of all wounds and infective diseases of the face and scalp, and of the brain and its membranes, when these are exposed by injury, disease, or operation. Chronic purulent disease of the ear should be regarded as a constant menace to the brain and its sinuses and envelopes.

DISEASES OF THE INTRACRANIAL VEINS.

Little is known about diseases of the intracranial veins. Venous hemorrhage, although comparatively infrequent, occurs sufficiently often to be worthy of some attention. Wiglesworth, as already stated, believes that some cases of dural hematoma originate from venous hemorrhages. The intracranial hemorrhages which occur in young children during the paroxysms of whooping cough are, in some instances at least, from ruptured veins. In death from asphyxia, particularly if suddenly produced, hemorrhage may take place from veins and capillaries. I have twice had the opportunity of examining the brains of men who have been hanged, in both cases within a very short time after execution, and numerous capillary and small venous extravasations were present in the floor of the fourth ventricle and elsewhere in the brain. Veins and sinuses are sometimes injured during surgical operations, causing hemorrhages. Occlusion of veins is much more common than hemorrhage. Throm-

bosis of both veins and sinuses may be coincident, as noted in the preceding section. Doubtless in many cases of sinus thrombosis the coagulæ extend into the veins. Clots form somewhat readily in the cerebral veins, because the outpour of blood from the arteries into them is for most of them in opposition to gravity. Localized thrombosis of a cerebral vein may give rise to such cortical symptoms as monospasm or unilateral convulsions followed by monoplegia or hemiplegia. Thrombosed veins have been found in the motor cortex in a few cases of this kind. Emboli from a distance may lodge in the cerebral veins; and phlebitis of the cerebral as of other veins is an occasional accompaniment of various infectious diseases. Arteriovenous aneurisms are sometimes met with, and may result in perforation of the skull. So little is known about the clinical history of these affections of the encephalic veins that nothing need be said about their diagnosis and treatment.

MALFORMATIONS OF THE BRAIN AND ITS ENVELOPES.

Certain abnormalities of structure of the brain are best considered in connection with diseases of the encephalic membranes, as these are often involved in the malformations produced. Neuraxial malformations in general would perhaps be best considered together, so that, strictly speaking, the spinal abnormalities of structure homologous with those of the encephalon might be treated of here;

Fig. 216.

Anencephalus. (Hirst and Piersol.)

Fig. 217.

Exencephalus. (Hirst and Piersol.)

but, as this method would probably disturb other points in the arrangement of the work, these affections will be discussed later. They include such diseases as *rachischisis* or *spina bifida*, which embraces spinal *meningocele*, *spina bifida occulta*, and *myelocele*. Arrests and aberrations of brain development include such abnormalities as *anencephalus* (Fig. 216), or absence of the brain; *exencephalus* (Fig. 217), or escape of the brain from the skull; *pseudencephalus*, where

the brain and its envelopes and vessels are represented by only a few rudiments; *cyclocephalus*, or *cyclopia*, where because of the fusion of the anterior cerebral vesicles of the two hemispheres the two orbits and eyes become merged into one, so that there is a single rudimentary eye; and *porencephalus*, or limited arrests of development shown by the absence of convolutions or lobes causing irregular subpial cavities. Porencephalus is frequent in the cerebral palsies of children, and will be considered more fully when these affections are discussed. Other abnormalities are *encephalocele* (Fig. 218), or the protrusion or hernia of brain substance through an opening in the skull which is usually in the median line; *hydrencephalocele*, a form of encephalocele containing fluid, and usually communicating with the ventricles; and *meningocele* (Fig. 222), protrusion from the cavity of the skull of a portion of the cerebral membranes, a pure meningocele being present when the tumor containing the fluid does not communicate with the interior of the brain. When brain substance and membranes are together extruded from the cranial cavity, the tumor and cyst thus formed constitute a *meningoencephalocele*, a pure encephalocele being present when the protruding brain substance ruptures and forces its way through the membranes as well as through the cranium. *Acrania*, or absence of the skull, does not necessarily include anencephaly. Such monstrosities as acrania, anencephalus, exencephalus, pseudencephalus, and cyclopia have, of course, no therapeutic interest. Limited arrests and aberrations may occur, such as absence of the fornix, of the callosum, and of special association tracts, trilobar and quadrilobar brain, and what is known as *inoccipitia*, or deficiency of the occipital lobe. Most of these malformations are of scientific rather than of practical interest. In chronic hydrocephalus and in meningocele something can be done by treatment towards prolonging life, and these affections will be considered in some detail. It may be important also to diagnosticate them from some of the curable or incurable organic diseases of the nervous system; as, for instance, in a case of infantile cerebral palsy to determine whether it is due to chronic hydrocephalus, to porencephalus, or to some other lesion, as hemorrhage, sclerosis, or tumor. *Microcephalus*, or narrowing of the brain in all directions, will be

Fig. 218.

Encephalocele. (Hirst and Piersol.)

more conveniently considered under idiocy and imbecility. In considering these malformations it is of first importance to remember the facts and theories which have been presented in the section on the development of the nervous system, and particularly those portions which relate to the cerebral vesicles. Embryonal arrests and aberrations of vesicular development account for most of them.

CHRONIC HYDROCEPHALUS.

Definition.—Chronic hydrocephalus is an affection in which an effusion slowly takes place into the ventricles (*internal hydrocephalus*), or subarachnoid spaces (*external hydrocephalus*). Various forms of acute hydrocephalus have been considered in connection with tubercular and serous meningitis. Occasionally cases of this kind arising acutely pursue a subacute or chronic course, but one which, as a rule, is comparatively short. In rare cases, as has been indicated under serous meningitis, this affection is practically cured, a small amount of hydrocephalic distention usually remaining.

Clinical History.—Chronic internal hydrocephalus, which is the most common form, while dating from birth or before, is not always recognized at that time, although occasionally the brain may be so large as to cause by its size interference with delivery. Infants congenitally hydrocephalic may be attacked with convulsions soon after birth, and to acute observers may appear abnormal or defective. In marked cases the head soon begins to enlarge; in others attention may not be directed to this enlargement for weeks, months, or even one or more years. In these the effusion is of very slow progress, and the disease is not infrequently attributed by the parents to a blow or fall or to some acute illness. When the enlargement of the head begins early, and sutures and fontanelles remain open and gradually widen, after a time the skull bears a striking disproportion to the face, sometimes giving the head and face a triangular appearance, the latter narrowing to a point at the chin (Fig. 221). Usually the skull becomes rounded or ellipsoidal, but occasional irregularities of the two sides of the head, or local irregularities, are observed, due to the manner in which local ossification has taken place. Sometimes the eyes are pressed downward because of changes in the orbital bones. The veins of the skin enlarge and become distorted, and the growth of hair is usually scanty. The child does not develop mentally, or, if at all, very slowly and irregularly; at an early age it seems to be lacking in powers of attention; it continues to be uncleanly, cannot be amused or corrected like other children, and after a time general attention is directed to its defective intelligence. It is sometimes unable to hold up its head, and is usually restless, peevish, and hard to amuse and control. Not infrequently it has attacks of spasm, or even a form of epileptic status, one seizure

succeeding another at short intervals. Strabismus is not uncommon. The power of standing and walking may not be acquired; or if the child learns to walk it is often at a late period and in an imperfect manner. In some instances, however, the body and limbs are symmetrical, and the patient is able to walk and move its upper extrem-

Fig. 219.

Chronic hydrocephalus with mental deficiency and epileptiform attacks; atrophic paralysis with contractures.

Fig. 220.

Chronic hydrocephalus with imbecility; paralytic and spastic conditions of the limbs are absent.

ities with ordinary freedom and skill. These are cases in which the pathological condition does not attain a high grade, but even these often show slowness, clumsiness, or uncertainty, and are nearly always very deficient mentally. Sight and hearing are usually preserved and may be comparatively acute. Apparent dulness of sight

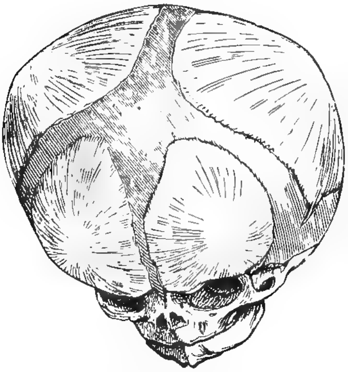
and of hearing may be due rather to mental weakness and apathy. Occasionally the head attains an enormous size, cases having been reported in which the head measured more than forty inches in circumference; and the dimensions may vary from this down to little or nothing above the normal. Now and then a hydrocephalic child seems to show unusual precocity, as in the case of one of the children at the Pennsylvania Training School for Feeble-Minded Children, who indulged in recitations, imitations of others, and grotesque profanity, which he was prone to exhibit on all occasions. Such appearances of intelligence are usually very superficial. The above description applies to cases which survive to at least a few years. Some die in convulsions soon after birth. Most cases die before puberty, although some live to middle life or very rarely to old age. I once made an autopsy on a case that had attained the age of nearly fifty years. In this case the head was not of unusual size, but the ventricles were enormously dilated. The man had preserved the full use of his limbs and possessed a fair degree of intelligence, and probably ranked as a high grade imbecile. The case shown in Fig. 219 is now about thirty years old, and presents marked atrophy with paresis and contractures of the right half of the body. He has convulsive paroxysms at irregular intervals. He is mentally deficient, and yet possesses considerable shrewdness and intelligence and is largely able to care for himself. The patient shown in Fig. 220 is of low grade mentally, but has not the atrophic and paralytic conditions presented by the preceding case, his face, trunk, and limbs being comparatively symmetrical.

Etiology.—Chronic hydrocephalus of considerable duration—of several years at least—may be associated with some forms of brain tumor in childhood, as with gliomata of the cerebellum. The growths in these cases, or at least in some of them, probably produce the hydrocephalus mechanically by pressure on the veins of Galen or by closure of the foramens of Magendie and Mierzejewski. The most common type of chronic hydrocephalus is congenital, and is due to defect of structure, alterations in the quality of the blood, or possibly in some cases to prenatal inflammation of the ventricular ependyma.

Pathology.—The bones of the cranial vault are usually much thinned and expanded, and are often widely separated at the sutures and fontanelles, as shown in Fig. 221. Wormian bones may be found in the interspaces between the usual bones constituting the skull, or in extreme cases the entire vault may present the appearance of islets of bone connected by tough membrane. Complete cranial ossification has, however, in some instances taken place, and rarely the skull may even be thickened. The lateral ventricles and their horns are, as a rule, enormously dilated, and sometimes the aqueduct, the fourth ventricle, and even the spinal canal may take part in the dila-

tation. In congenital noninflammatory cases the fluid differs little, if at all, from the normal cerebrospinal liquid. In acquired cases it is sometimes turbid or flocculent, and may even contain a very small quantity of pus or blood. In these cases, also, the membranes at the base, the choroid plexus, and the ependyma of the ventricles may be granular or otherwise changed. The fissures and gyres are largely obliterated; the pia is usually thin, and the surface of the cerebrum smooth. Structural changes are found in both the gray and the white substance, and yet they often preserve their functions to a remarkable degree, considering the amount of compression which is exhibited.

FIG. 221.



Skull of a hydrocephalic infant. (Agnew.)

Diagnosis.—The diagnosis may be difficult until the peculiar changes in the conformation of the head have begun to take place. It will then be based upon a study of these changes and of the symptomatology of the disease already given. Occasionally the heads of rachitic infants bear a considerable resemblance to those of hydrocephalics; but the differentiation is here made by a study of the other physical conditions commonly present in rachitics, such as changes in complexion and general appearance, softness and deformities of the ribs or of the bones of the extremities, and enlargement of the liver. The rachitic head is squarer in shape than the hydrocephalic, the fontanelles are not so protrusive. Hypertrophy of the brain with coincident enlargement of the skull is a rare affection, which perhaps might be confused with hydrocephalus; but in the former the fontanelles and sutures are unlike those in a case of hydrocephalus, and the general conformation of the head and face is different. Certain rare osseous dystrophies cause thickening of the cranial walls, and consequent enlargement of the head; these may be difficult to distinguish from hydrocephalic enlargements. A peculiar percussion note due to thinness of the skull and brain walls is given in hydrocephalus, and this may assist in distinguishing the affection from enlargement of the head due to thickness of the bones. It is difficult and almost impossible during life to distinguish between internal and external hydrocephalus. A very carefully made puncture might show that effusion was subarachnoidal, but it must be remembered that only a few millimetres separate the subarachnoidal space from the enlarged ventricular cavities.

Prognosis.—The prognosis of chronic hydrocephalus is very

unfavorable. A few cases make spontaneous partial recoveries; and a few make slight temporary improvement under careful surgical treatment.

Treatment.—The medical treatment of chronic hydrocephalus is scarcely worthy of discussion. Cathartics and diuretics can only temporarily affect the intracranial contents; and absorbents, such as mercury and the iodides, while theoretically indicated, can do little or no good, as they do not reach the continuing cause of the disease. Tonics and nutrients may improve the general condition of the patient. The methods employed by surgeons have been directed to the relief of hydrocephalus by evacuation of fluid, either alone or in association with compression. The most common method of evacuation has been directly through the cranial walls or through the fontanelles or sutures. The puncture can be made at almost any position, particularly in cases of great enlargement; but Keen and others have advised special points for puncture. Lumbar puncture has been suggested by Quincke and others. Evacuation of fluid without compression avails nothing, as the fluid rapidly reforms. Even with compression in connection with repeated punctures little can, as a rule, be gained. Compression can be made by strips of diachylon plaster or any strong adhesive plaster, or by elastic bands.

MENINGOCELE.

General Considerations.—Meningocele may be either encephalic or spinal, but both forms have practically the same pathology. In encephalic meningocele a tumor is formed outside of the skull, by the escape of the arachnopia covered by the dura. The translucent vesicle thus formed, which may be of varying size, communicates by a narrow neck with the subarachnoid cavity, and is filled, therefore, with cerebrospinal fluid. It is usually in the median line and in the occipital or nasofrontal region.

FIG. 222.

Meningocele. (Graham.)

Clinical History.—In true meningocele the tumor is usually observed immediately after birth. It is commonly more or less rounded in shape, and the defect in the skull can often be felt. Crying and laughing or any violent expiratory effort increases the tension of the tumor. The fontanelles are usually large and the sutures open. Various paralytic defects and deformities of the face and limbs may be present. The tumor, which is sometimes

sensitive at first, gradually increases in size. Compression may cause convulsions and bulging of the anterior fontanelle and frontal bones; occasionally the tumor is divided into two or more lobes. As a rule, the cases live only a few days, weeks, or months.

Etiology and Pathology.—Congenital meningocele is due commonly to embryonal arrest of development. Intrauterine hydrocephalus is probably present in most cases. The pathological appearances, so far as known, are those already given in the general description of the affection.

Diagnosis.—Meningocele has been mistaken for brain hernia, vascular growths, sebaceous cysts, and even for abscesses. According to Weinlechner, the diagnosis can be readily made if the tumor is partly reducible; if it pulsates synchronously with the heart; if it increases in size upon crying; if an opening in the bone can be felt, and if cerebral disturbance follows pressure. When these signs are absent, the withdrawal of a small quantity of the fluid by means of a hypodermatic syringe will by determining that this is cerebrospinal fluid demonstrate its intracranial origin. An encephalocele is usually opaque and has a broader base, while a meningocele and a hydrencephalocele are generally translucent and pedunculated. Cranial fracture with laceration of the meninges and cerebrospinal effusion and possible communication with one of the lateral ventricles may give rise to a spurious meningocele known also as acquired cephalocele or traumatic cephalohydrocele. In such a case the traumatic origin is shown by a history of injury and the appearance of the tumor at a point other than one of the sutures. Dr. Francis Huber collected over twenty such cases. According to Weinlechner, quoted by Huber, the tumor may appear immediately after injury, or may be delayed several weeks or months. Pressure in such cases may cause no cerebral disturbance, and pulsation is usually absent. (Graham.)

Prognosis.—The prognosis is almost always very grave, death occurring in a short time. Sometimes the tumor bursts after attaining considerable dimensions. In rare instances, when the cleft and enlargement are small, closure takes place and recovery follows. In rare instances also surgical interference is successful.

Treatment.—To protect the tumor from external pressure or injury, and with the hope of limiting or checking its growth, pads or covers can be used. The withdrawal of fluid with antiseptic precautions and the application of pressure, or puncture followed by the injection of a solution of iodine ten grains, potassium iodide thirty grains, and glycerin one fluidounce, has been followed by cure. Operative treatment is much more likely to be successful when communication with the cavity of the cranium no longer exists. The radical operation of removing the sac and closing the wound, with antiseptic precautions, has been successfully performed.

CHAPTER IV.

ENCEPHALIC HISTOLOGY AND PHYSIOLOGY IN THEIR RELATIONS TO FOCAL DISEASES OF THE BRAIN.

GENERAL CONSIDERATIONS.

THE best method of discussing focal diseases of the brain is one based in a general way on its embryonic subdivisions, modified considerably by the requirements of practice,—by what experience has taught to be the most frequent sites and extensions of lesions. Hemorrhage, softening, tumor, or abscess may be limited to an embryonal subdivision, but on the other hand the same lesion may invade two or more parts. Cortical focal diseases are, as a rule, confined to the cortex and subcortex of the prosencephalon, but deep seated lesions commonly invade both prosencephalon and thalamencephalon, as where intracerebral hemorrhages and softenings partly destroy striata, capsules, and thalamus together. Other parts entering into the formation of the forebrain—as the olfactory bulb, callosum, fornix, optic nerve, hypophysis, and conarium—are often the foci of disease or the seats of special processes of degeneration. The affections of the midbrain—which includes the iter, quadrigeminal body, and crura—are probably best considered apart or with those of the pons. Focal diseases of the cerebellum are probably also best considered separately, although the pons and cerebellum together are derived from the fourth secondary vesicle, and hence jointly constitute an embryonic subdivision. Diseases of the pons and oblongata can be discussed in part separately and in part together. Usually diseases of the cranial nerves are considered in a special chapter or chapters, and it is best perhaps to follow this method. The difficulties which beset any method are, however, great, as is illustrated by the fact that in the diagnosis of symptoms which may be dependent upon lesions of the cranial nerves—as hemianopsia or paralysis of ocular movements—it may be necessary to take into consideration affections of the mesencephalon, thalamencephalon, or prosencephalon. The focal diseases of the brain include hemorrhage, embolism, thrombosis, aneurism, tumor, and abscess; and, in the next chapter, it will be found most practical to consider these focal diseases and some of their most important consequences, such as monoplegia, hemiplegia, hemianesthesia, monospasm, athetosis, aphasia, and hemianopsia; and also some of the cerebral palsies of children, which may be due to arrests or destructive lesions of the forebrain, presenting characteristics dependent upon the time

and manner of their development. Postmortem examinations of the brain, especially with reference to the localizing and recording of lesions, are best considered in connection with the study of focal diseases, as is also localization in its relations to craniocerebral surgery. The general anatomy and general physiology of the brain, as of other parts of the nervous system, having already been considered, it will only be necessary to discuss briefly the physiology of special regions before considering their focal diseases. This discussion will involve a summary of the minute anatomy of cortical and other structures, and a presentation of accepted facts regarding encephalic localization.

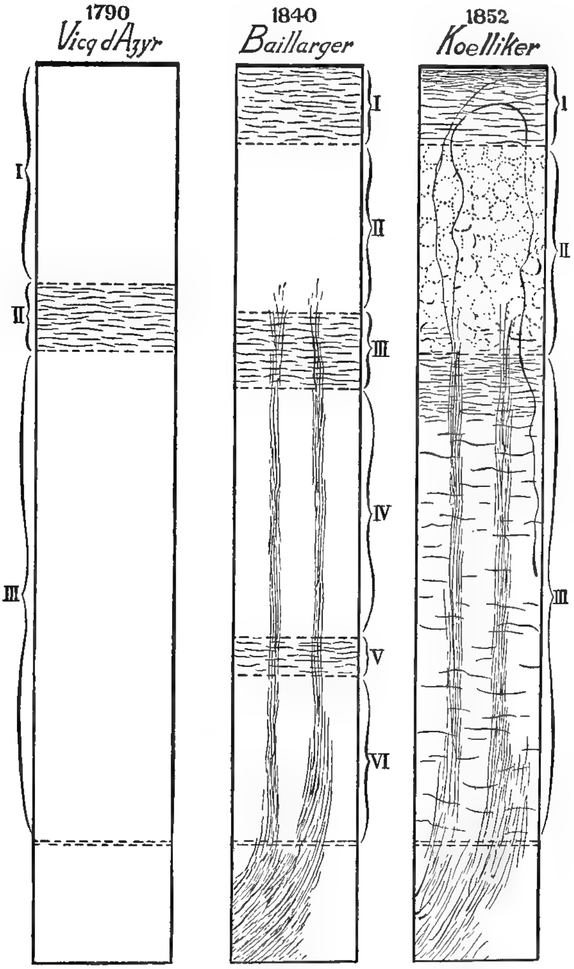
MINUTE ANATOMY OF THE CEREBRAL CORTEX.

General Features of the Cortex.—The depth of the cortex, or rind of the cerebrum, is usually reckoned as from two to three millimetres, but in health it may vary from one and two-tenths to four millimetres. The greatest depth is at the summits of the convolutions; regionally its maximum depth is in the mesal portion of the so-called motor region. It diminishes in depth backward from this region to the occipital pole, but all gross determinations of the thickness of the cortex in average normal brains are liable to error. The depth of the cortex of the right hemicerebrum is stated to be slightly less than that of the left, and the same is said to be true of the female as compared with the male brain. Various arrests and diseases may cause great variations of cortical thickness. The superficial extent of the cortex has also been shown by ingeniously contrived and executed methods of measurement to vary considerably in health, and sometimes enormously in disease. The specific weight of the cerebral cinerea, whether cortical or ganglionic, is somewhat less than that of the white substance. A vertical section of the cortex, particularly in certain locations, can be seen even with the naked eye to have a laminated arrangement.

Histological Studies.—Since the wonderful discoveries of Golgi, whose methods have been adopted and improved upon by numerous investigators, intricacies of cell structure not dreamed of a few years since have been revealed, resulting in the general recognition of the "neuron" or nerve cell as the unit of nervous and psychical function, and of the fact, now universally conceded, that these nerve cells transmit impulses by contact of their processes and bodies. We are therefore now able to apply more clearly to the solution of physiological and pathological problems the truths determined by histology. Knowing the origin, course, and methods of association and communication between these nerve cells, we gain a clearer conception of the phenomena which are still properly referred to special areas of the cortex or to subcortical ganglia and

tracts, and also of the effects of disease and dissolution upon cerebral structures. According to the views which prevailed down to the time of Golgi, the connections between neighboring nerve cells were supposed to be affected either by anastomosis of cells and processes or by fusion of other processes into a finely granular or

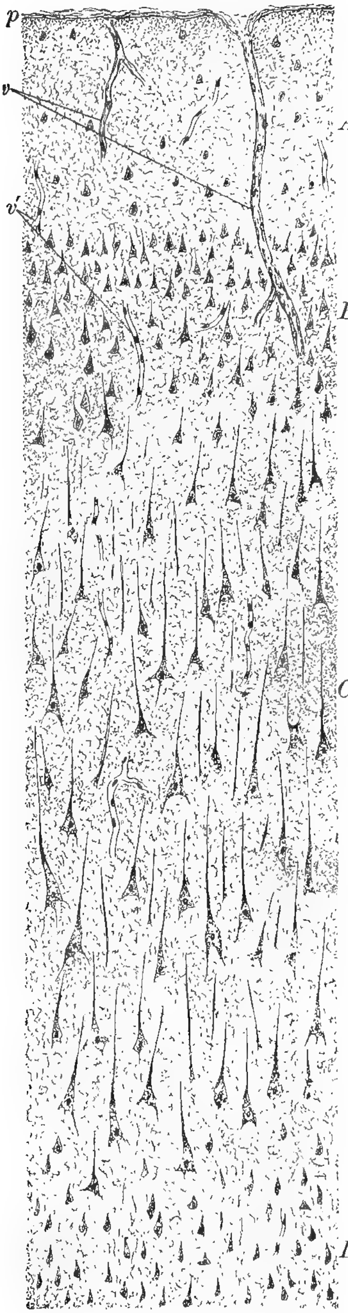
FIG. 223.



Diagrams indicating the views of Vicq d'Azyr and Baillarger, and the earlier views of Koelliker, as to cortical lamination. (After Dejerine.)

amorphous mass, called the granular substance. The stains and methods of microscopical investigations employed were unequal to the task of unsealing and unravelling the mysteries of this substance and the apparently anastomosing nerve fibres. Not only have views undergone a radical change with reference to the nature of formerly undemonstrated structures and the methods of cell communication,

FIG. 224.



Section of human cerebral cortex stained with sodium carminate: A, B, C, D, first, second, third, and fourth layers; p, pial tissue; v, v′, bloodvessels. (Piersol.)

but it has become necessary to modify other opinions, such as those relating to cortical lamination, and the methods of entrance and exit of numerous systems of nerve fibres into the various layers of the cortex.

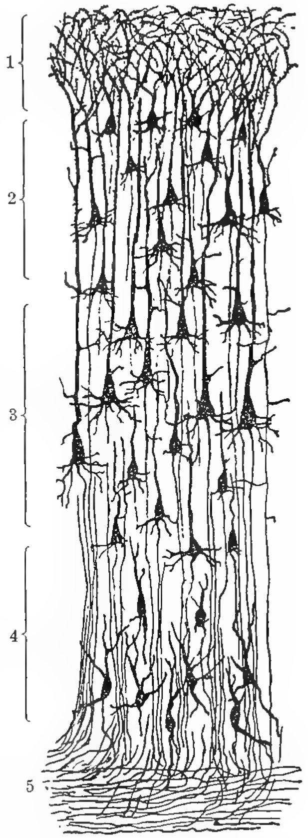
Discoveries of Golgi.—Golgi showed (1) that the protoplasmic expanses and branchings of the nerve cells preserved their individuality to their free terminations; (2) that branching processes of adjacent cells did not anastomose; (3) that processes did not blend and fuse into a granular basic substance; (4) that, in addition to the basic protoplasmic processes, the pyramidal, the fusiform, and the polymorphic cells gave off axis cylinder processes which in their turn had processes branching at right angles to their general course; (5) that a plexus was formed by fine fibrils or collaterals ramifying into other fibrils of extreme delicacy; (6) that some cortical nerve cells had long axis cylinder processes which preserved their individuality for a great distance and became medullated nerve fibres of the white substance, and that others had short axis cylinder processes which lost their individuality in the immediate neighborhood of the cells from which they sprang. He regarded the first as motor and the second as sensory cells.*

Cortical Lamination.—Vicq d'Azyr regarded the cortex as constituted of only three layers. Baillarger held to six, arranged from

* In the preparation of this section I have made free use of the contributions of W. Lloyd Andriezen, and especially of his article on Some of the Newer Aspects of the Pathology of Insanity, in Brain, Part IV., 1894, pp. 548–692.

within outward in layers of alternating white and gray matter. Koelliker in 1852 consolidated these layers into three. The views of these authors are indicated diagrammatically in Fig. 223. With improved microscopical methods new subdivisions of different regions of the cortex were made by Meynert, Bevan Lewis, Mierzejewski, Clarke, and others. The Rolandic cortex has been most studied as regards lamination and other structural peculiarities, and the descriptions and diagrams of recent years have been based chiefly upon investigations of this region. Its subdivision by Meynert into five layers is the one that has been generally given in textbooks, treatises, and monographs, sometimes with slight modifications in detail. Four of these five layers are shown in the diagram, Fig. 224. The lamination of the cortex, however, differs in different regions of the brain, as especially shown by Meynert and Bevan Lewis. Altogether, Meynert has described five regional types: (1) a common cortical type; (2) an occipital type; (3) a Sylvian type; (4) a type belonging to the cornu ammonis; and (5) a type peculiar to the olfactory bulb. The investigations in regional variations of lamination by Meynert and Bevan Lewis, although of great interest and value, cannot here be given. The five layers as usually given, following Meynert, were made up of (1) an outer or superficial layer of basis substance and neuroglia, long thought to contain no nerve cells, but in which Exner later discovered an intricate nerve plexus; (2) a clearly defined layer of closely packed small pyramidal cells with their apices towards the brain surface, and axis cylinder processes passing inward; (3) a layer chiefly of large pyramidal cells interspersed with small pyramids, often called the formation of the cornu ammonis, because most strikingly developed in this region; (4) the granular formation of Meynert, composed chiefly of small, rounded, angular nerve cells and also radiating medullated nerve fibres; (5) the claustral formation of Meynert, a layer composed chiefly of fusiform or spindle-shaped cells interspersed with rather large but

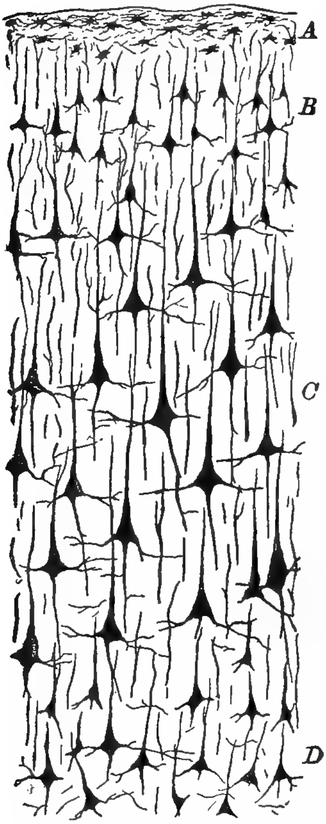
Fig. 225.



Perpendicular section of the gray substance of a cerebral convolution: 1, molecular layer; 2, layer of small pyramidal cells; 3, layer of large pyramidal cells; 4, layer of polymorphic cells; 5, white substance. (Ramón y Cajal.)

short pyramidal cells. Bevan Lewis also described five layers as peculiar to the motor or Rolandic cortex, but did not separate them exactly after the manner of Meynert. Golgi recognized three layers as existing everywhere in the cortex, although these are not sharply defined in all locations, and also described three great types of cells, —the fusiform, the pyramidal, and the globular or polygonal. The pyramidal cells, according to him, were mainly in the superficial and middle layers, the globular cells occurring everywhere, but most abundantly in the neighborhood of the fusiform or spindle-shaped cells, which were found almost entirely in the deepest of his three layers.

Fig. 226.



Section of cerebral cortex (motor area) of a child, stained by Golgi's silver method: *A*, layer of neuroglia cells; *B*, layer of small pyramidal ganglion cells; *C*, layer of large pyramidal cells; *D*, layer of irregular smaller cells. (Piersol.)

Subdivision of the Cortex into Four Layers.—Following Ramón y Cajal, the now generally accepted division of the cortex is into four layers: (1) a molecular layer; (2) a layer containing small pyramidal cells of variable shape, called by Andriezen the ambiguous; (3) a layer of large pyramidal cells with long apical processes; and (4) a polymorphic layer. This method of subdivision into four layers is shown in Figs. 225 and 226. In some cortical regions striking departures from the usual arrangement into these four layers are found. In the cornu ammonis, for instance, the molecular layer and the large pyramidal cells are conspicuous, the ambiguous layer being practically absent.

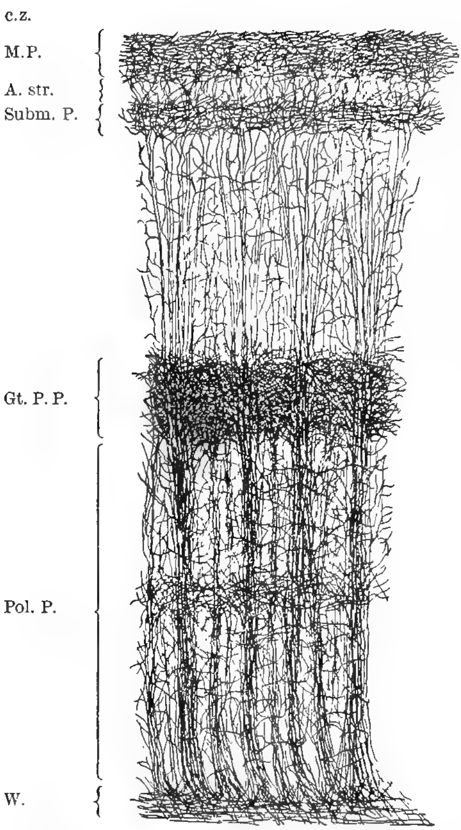
Types of Cells in the Four Cortical Layers.—Eight types of cells are now recognized as entering into the composition of the four cortical layers: (1) pyramidal cells with long ascending apical processes which reach the molecular layer; (2) pyramidal cells with short apical processes which do not reach the molecular layer; (3) ambiguous cells which have various subtypes, as the globose, fusiform, and asymmetrical bicornute; (4) granule cells with short and imperfect protoplasmic processes; (5) fusiform or triangular cells with ascending axis cylinder processes, sometimes called Martinotti's cells; (6) fusiform cells with descending axis cylinder processes;

(7) oblique and inverted pyramidal cells with descending axis cylinder processes; (8) polygonal cells with short branching axis cylinder processes, sometimes spoken of as Golgi's sensitive cells. Other cells, fusiform and triangular, have been described by Ramón y Cajal as present in the molecular layer, and were regarded by him as embryonic. Andriezen has not succeeded in discovering these in man. The behavior of these cells in the presence of staining agents is, of course, a matter of vital importance in their study. Some, like the large pyramidal cells, are *chromophilous*, staining readily; others are *chromophobic*, staining with difficulty. Among the latter are the triangular, the polygonal, and some fusiform cells. Various gradations in their reactions to stains are found throughout the list of cells.

The Molecular Layer.—The outermost portion of the molecular layer, the most superficial film of the cerebral substance, is composed of a system of neuroglia fibre cells called by Andriezen the *caudate* cells. These cells and their processes form a fine feltwork of cortical glia fibres. A few other glia cells are found in the molecular layer. They unite the caudate cells with stellate fibre cells which are abundant in the alba and are sparsely present in the cinerea. Next below are the neuroprotoplasmic structures, formerly classed under the general term molecular substance. The following structures have been determined as entering into the composition of this molecular substance: (1) descending neuroglia fibres from the caudate cells; (2) transitional fibre cells and their processes; (3) a forest of protoplasmic processes ascending and branching from the ambiguous layer; (4) protoplasmic processes of tuftlike appearance ascending from the long pyramidal cells; (5) cell bodies and protoplasmic processes of polygonal cells intrinsic to the molecular layer (Golgi's sensitive cells); (6) special protoplasmic glia cells with their processes, found in this and in other layers of the cortex and first described by Andriezen; (7) Exner's plexus—nerve fibres of various kinds, including axis cylinder processes, some of which have been traced from the centrum ovale and which are probably either association, commissural, or projection fibres—intermixed with which is a larger number of naked processes, collaterals and terminals; (8) fine, non-medullated, terminal branches and collaterals derived from the thick fibres of the last system described, either as offshoots from below or from the peripheral branches in the molecular layer itself; (9) ascending axis cylinders from fusiform, triangular, and polygonal cells in the deeper cortex; (10) branching short axis cylinders from deeply situated local polygonal cells; (11) ascending collaterals from the axis cylinders of the subjacent pyramidal cells; (12) a few ascending collaterals from Golgi's polygonal cells in or below the ambiguous layer; (13) certain fusiform nerve cells, described by Ramón y Cajal, in the

upper part of the molecular layer. This single layer of the cortex is thus seen to contain a wilderness of nerve fibres, but a wilderness which has proved penetrable to modern investigation. This molecular layer, especially in certain regions, as that of the cornu ammonis, has been found to subdivide readily into three strata, which from without inward are termed (1) the *stratum moleculare*, (2) the *stratum lacunosum* (corresponding to Exner's plexus), and (3) the *stratum radiatum*. A glance at Fig. 227, by Andriezen, which is intended to represent the nerve fibre systems and plexuses of the entire cortex, shows the extraordinary complexity of these structures.

Fig. 227.



Cortex of human brain, showing the nerve fibre systems and plexuses (combined Weigert's and Golgi's methods): c.z., clear zone (free of nerve fibre); M.P., molecular plexus (Exner's), in the molecular layer; A. str., ambiguous cell stratum; Subm. P., submolecular plexus; Gt. P.P., great pyramidal plexus; Pol. P., polymorphic plexus; W., white substance. (W. Lloyd Andriezen.)

The Ambiguous Layer.—To the second layer of the cortex Andriezen has applied the term *ambiguous*, because of the variability and indefinite shape of many of its cells. They are chiefly fusiform, pyriform, triangular, or polygonal, with many small pyramidal cells interspersed, and hence the formation was called by Ramón y Cajal the layer of small pyramidal cells. One striking feature of the cells of this layer is their bifurcating thick upper part, giving the appearance of two unequal horns. These cells, which are chromophobic, are enormously developed on the posteroinferior portion of the hippocampal gyre. They have distinct axis cylinder processes which usually come off obliquely and descend through the subjacent laminæ. Some of these processes are in the deeper parts of the cortex, while others have not been traced to their destination. On the confines of this layer, both above and below, the so-called Golgi's sensitive cells appear, and branch rapidly into a meshwork of terminal fibres.

The Layer of Large Pyramidal Cells.—The layer of large pyramidal cells, often called the formation of the cornu ammonis, because of its enormous development in that region, is one of vast importance in the study of the cellular physiology of the nervous system. The pyramidal cells with short apical processes are found everywhere in the cortex below the molecular layer. Their terminations never pass into this layer, but end in the ambiguous layer. They are regarded by Andriezen as belonging to a different system from the pyramidal cells with long apical processes. The layer of these large pyramidal cells is, however, more definite in position and connections; the cell processes invariably reach upward and end in the molecular layer—mainly in Exner's plexus. These apical processes also reach to some extent into the molecular stratum, and send lateral branches into the radiate stratum or sublayer. The cell bodies increase in size as the layer is descended,—that is, as the polymorphic formation is approached. A basilar system of protoplasmic processes extends downward, ending in the polymorphic layer. Andriezen has shown that both the apical and the basilar protoplasmic processes have definitely traceable connections. These are (1) the apical processes with naked collaterals and terminals of the medullated nerve fibres which ascend from the white substance and ramify in Exner's plexus; the terminals and collaterals of the medullated fibres also come into contact with the processes of the ambiguous cells when these latter are present; (2) the basilar protoplasmic processes with a system of fine medullated nerve fibres—whose terminals and collaterals are, however, nonmedullated—which ascend from the alba. Thus are formed basilar and apical nervoprotoplasmic plexuses. While the vast majority of the fibres from the white substance end in the lacunar stratum of the molecular layer (Exner's plexus), a much smaller number terminates in its other strata. Thus are constituted the great *extrinsic* connections of these cells, which, however, have also *intrinsic* relations with the adjacent cells. One other important connection of the apical processes of the large pyramidal cells is with the axis cylinder processes of cells in the polymorphic layer, called Martinotti's cells, which are usually fusiform or triangular. The intrinsic cells come in contact with the pyramidal cells by terminal tufts of fibrils which clasp the bodies of these cells. These last described connections have been traced only in the regions of the cornu ammonis and fascia dentata. Certain nerve fibres leave the body of the pyramidal cell at its base and pass downward through the polymorphic layer to enter the white substance, giving off a few delicate collaterals at right angles in the subpyramidal layer. These constitute the beginnings of the motor system of fibres.

The Polymorphic Layer.—The main cell types in the polymorphic layer are described by Andriezen as (1) pyramidal cells

with short apical processes; (2) granule cells with short and imperfect protoplasmic processes; (3) fusiform cells with ascending axis cylinder processes (Martinotti's cells); (4) fusiform cells with descending axis cylinder processes which pass into the white substance; (5) asymmetrical or oblique pyramidal cells intermediate in form between the fusiform and the true pyramidal cells; (6) inverted pyramidal cells; (7) polygonal cells with short branching axis cylinder processes (so-called Golgi's sensitive cells). Andriezen regards the polymorphic system as a new development. The brain of the mammal possesses this layer, which is not found in the reptilian or amphibian brain, and which in the higher mammalian brains is intricately involved and complicated. Step by step this polymorphic layer develops in the higher order of mammalian brains. Differing from Meynert and Obersteiner, Andriezen regards the cells of this polymorphic system as having been originally pyramidal. It would seem that in the process of evolution, as more and more associations have been made, cells tending originally to be pyramidal have been twisted out of shape and position in this layer. The polymorphic cells have to a certain extent migratory powers. They are not the only association cells. The older systems of association fibres in the amphibian and particularly in the reptilian brain are found in the upper two layers of the cortex, and the callosum appears before the mammalian brain is reached.

Cortical Termini of the Sensory Projection System.—The sensory excitations passing upward to the cortex by projection fibres are distributed mainly in the molecular and submolecular regions, the impulses affecting the processes of both the ambiguous and the long pyramidal cells. This has been shown to be true of the olfactory and optic projection fibres, and is probably true also of the auditory and fillet radiations. Andriezen found that the olfactory radiations end chiefly in three places: (1) in the callosal portion of the genu of the gyrus fornicatus; (2) in the septum lucidum; and (3) in the inferior ends of the hippocampal and uncinate gyres. The optic radiations were found to terminate chiefly in accordance with the views of Henschen,—namely, in the cortex of the calcarine fissure. With others, he believes that the fillet radiations have their endings in the posteroparietal region, though a few of the fibres terminate cephalad of the central fissure. The entire cerebrum is regarded as an upward development of the sensory projection systems. The fillet system includes fibres for the transmission of tactile, pathic, thermal, muscular, gustatory, and perhaps other forms of sensibility. The olfactory and optic projection systems terminate in special areas of the cortex,—those which are usually recognized as zones or centres for smell and vision. Similarly the auditory radiations probably terminate in the superior temporal convolutions. In the molecular layer the terminal processes of the

sensory projection systems come into contact with the apical processes of the cells of the ambiguous and pyramidal layers. For this reason, according to Andriezen, the pyramidal and ambiguous cells should be regarded as the first sensory cells of the cortex.

Separate Sensory and Motor Localization.—The different theories as to the separate cortical localization of movements and of cutaneous and muscular sensation which have led to so much controversy have again become prominent in the light of the new researches just summarized. Those who contend against the doctrine that the Rolandic cortex is a purely motor region believe they have received additional support for their views. Let us recall here some of the varying hypotheses. Schiff holds that this region is the seat of tactile sensibility; Munk, that it is a general sensory area; Nothnagel and Hitzig, that it is related to the so-called muscular sense; Bastian, that it is kinesthetic, the only true motor centres being bulbospinal; others, in a more or less indefinite manner, that it is a sensorimotor region; while the view of Ferrier, Charcot, and many others is that the area is a true motor zone, one containing centres of movements which involve conscious discrimination. According to Horsley, in this region are represented tactile sensations in slight degree, the so-called muscular sense, and, preeminently, specialized movements. Some, like Waller, avoid the use of the terms motor and sensory centres, because this might imply spontaneity, holding that every centre must be a point to which impulses come as well as one from which they go. Bechterew holds that the motor and sensory centres are quite independent, but that they lie very close to each other and sometimes one over the other. As shown by Forel and Nansen, we have been too long handicapped by prevailing ideas of cell action and by theories of the parts played by the cell bodies as originating centres. Impulses are transmitted and transferred by processes as well as by the cell bodies. The function of the latter is chiefly trophic. The new researches and theories do not compel an abandonment of former views as to special localizations, although different standpoints may need to be taken. As already stated, the use of the term centre in neurology is largely one of convenience. Disregarding theory entirely, the subdivision of the cerebrum into physiological lobes, as indicated in Figs. 47 and 48, on pages 36 and 37, for practical purposes remains a good one. While the whole cortex in some of its strata may be regarded as a great sensory expanse, its Rolandic portion and particularly the convolutions cephalad to the central fissure constitute a region which is related to specialized movements of various parts of the body. One calls it motor, another kinesthetic, another sensorimotor, and another executive. For the purposes of the physician and surgeon it is a motor sphere. Its irritation causes specialized movements; its destruction impairs or abolishes these movements. It is as much

the area where the motor portion of the great sensorimotor act begins as where the sensory portion of it ends. The cerebral sensory area, cortical and subcortical, would from this point of view be that part of the cerebrum where the fillet radiations in their most compact form are nearest to the surface of the brain; and therefore this region might continue to be described as in the posteroparietal, quadrate, and fornicate convolutions. Destruction of this region causes loss or impairment of sensation, while irritation of it gives rise to phenomena of sensory excitement.*

CORTICAL LOCALIZATION.

The Prefrontal Lobes.—In the diagrams Figs. 228 and 229 the prefrontal region is termed "higher psychical," a not altogether unobjectionable designation. It is the area entirely cephalad of that universally recognized as related to the representation of movement. All parts of the brain in some way take part in mentation,—the brain in its entirety is the great psychical organ,—but this particular portion of the cerebrum is related to the highest of the high processes which have become through evolution the especial attributes of man. Hitzig, Ferrier, Goltz, Munk, Horsley and Schäfer, and Bianchi, among others, have experimentally studied the prefrontal lobes. These investigators have found more or less mental degradation to be the result of ablation or partial ablation of these lobes, the animals losing the faculty of close attention and intelligent observation; and undoubtedly impairment and disturbances of a peculiar character occur both in the lower animals and in man from lesions of this portion of the brain. The higher and more complicated intellectual processes—those which involve such faculties as attention, judgment, and comparison—are always affected. Inhibition is impaired. Bianchi extirpated one prefrontal lobe, and in some instances both lobes, in the dog and ape, and according to him these

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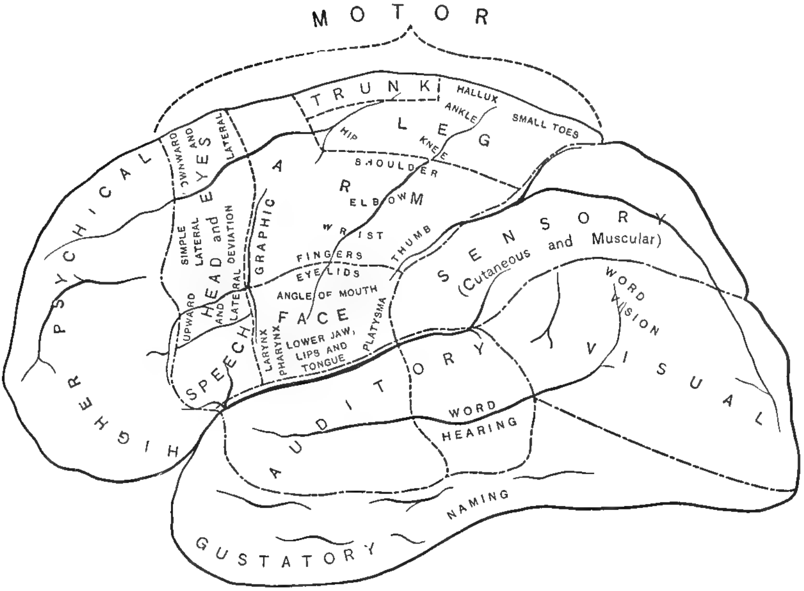
* **Flechsig's Subdivision of the Cortex.**—In this connection the most recent views of Flechsig should also be given. He makes two great divisions of the human convolutions. The one includes those areas which receive or give origin to the sensory or motor fibres (optic radiations, pyramidal tracts, fillet, etc.). The first area Flechsig refers to as "sensory centres." They include the areas of vision. The second division has no direct communication with the corona radiata, and contains only association fibres, the callosum and commissural fibres especially. The areas of the second great class, "association centres," occupy those portions of the brain that have not been allotted specific function by the localizationists. In the brain of a child three months old almost the entire corona radiata is medullated, and the streams of medullated fibres radiate to sensory areas. The association areas comprise four great tracts, the anterior portion of the frontal lobe, the island of Reil, the precuneus, and the posterior portion of the parietal lobe. It is probable that at this early stage of development each sensory centre possesses its own sensory mechanism, distinct from every other.

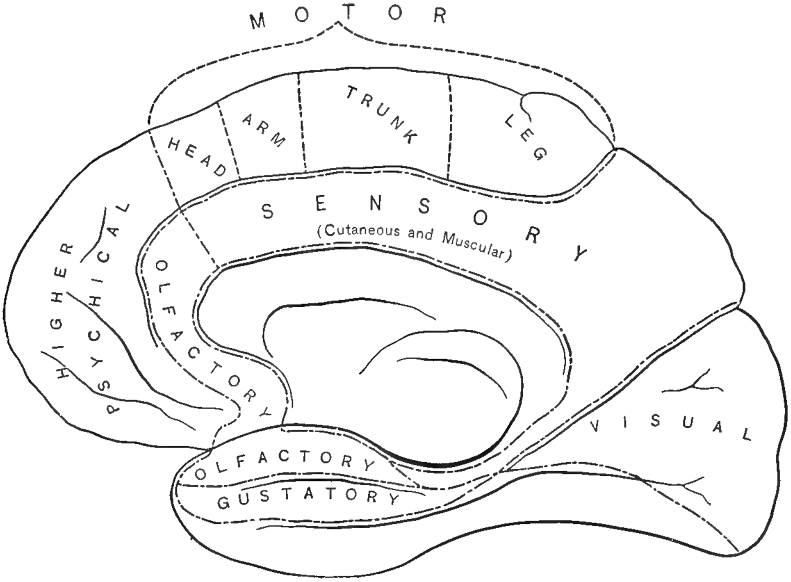
FIG. 228.



Zones and centres of the lateral aspect of the human cerebrum.

lobes are the organs in which the sensorial and motor products of the cortical zones are coordinated and synthesized. As stated on page 37, this region might be designated the lobe of re-representation or of

FIG. 229.

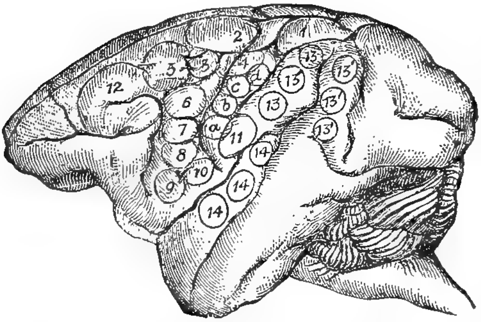


Zones and centres of the mesal aspect of the human cerebrum.

complex coordination. Bianchi believes that these lobes elaborate from one side the products of the cortical nerve cells of sense and motion in series, and from the other all the emotional states that accompany single perception, from the fusion of which arises "psychical tone." Destruction of these lobes causes disintegration of the personality, and incapacity to form serially groups of images or representations, more or less psychical dissolution occurring according to the extent of the lesion. Hesitation, uncertainty, fear, lack of force, weakness of the highest faculties, and motor inquietude due to loss of control, may be present.

Subdivisions of the Rolandic or Motor Cortex.—The divisions of the Rolandic or so-called motor cortex most in accord with known facts are shown in Figs. 228, 229, and 230. The region for the lower extremity is in the upper or most mesal portion, and is narrower than the other areas. The area for the upper extremity is in the middle portion of this region, and that for the face in the lower. The imaginary horizontal subdivisions are nearly in a line with the superfrontal and medifrontal fissures. Cephalad the region reaches a little forward of an imaginary extension of a line from the ascending branch of the Sylvian fissure to the edge of the hemisphere. Caudad it is bounded by the retrocentral fissure, although probably sometimes extending somewhat backward towards the parieto-occipital fissure. Some authorities include the superior parietal convolution in the lateral motor cortex; but it is more probable that this region belongs to the cortical sensory sphere (fillet radiations), or that the sensory and motor zones here blend or digitate. On the mesal aspect of the hemisphere the motor cortex includes the marginal convolution from the caudal termination of the callosomarginal fissure forward to the knee of the gyrus fornicatus, having from before backward head, arm, trunk, and leg subdivisions as shown in Figs. 229 and 231.

FIG. 230.

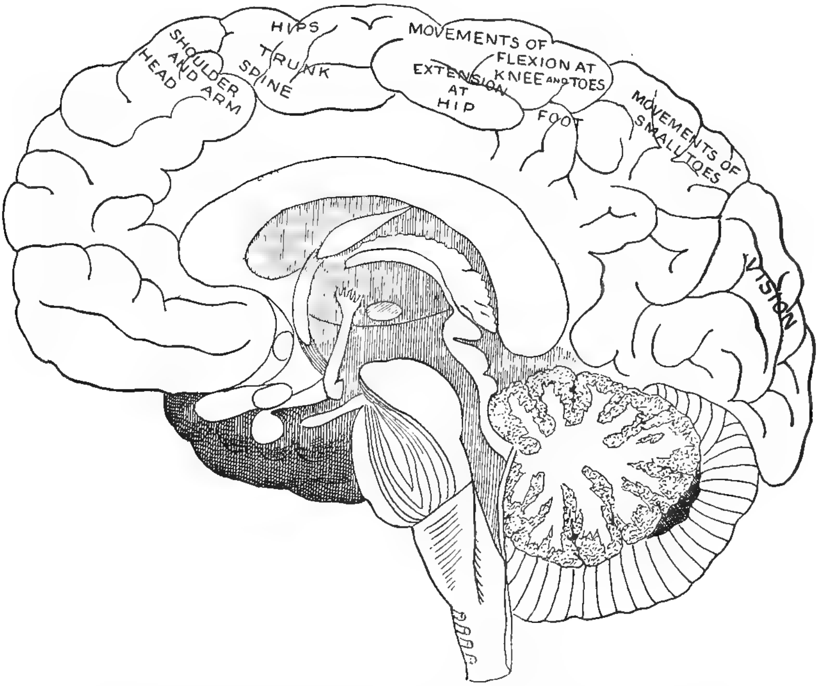


Lateral surface of the brain of a monkey: 1, centres of movements of the opposite leg and foot such as are concerned in locomotion; 2, 3, 4, centres for the various complex movements of the legs and arms; 5, centre for extension forward of arm and hand; 6, centre for movement of the hand and forearm in which the biceps is particularly engaged; 7, 8, centres for the elevators and depressors of the angle of the mouth respectively; 9, 10, orolingual centres; 11, centre of the platysma; 12, centre for lateral movements of the head and eyes with elevation of the eyelids and dilatation of the pupil; *a*, *b*, *c*, *d*, centres of movements of the fingers and wrists; circles 13, 13′, visual centres, including also occipital lobes; circles 14, auditory centres. (Ferrier.)

Subareas for the Lower Extremity.—While the cortical

representation for different segments of the lower extremity has not been worked out as accurately as for the upper, it has been determined that movements of the hip and knee are localized forward near the centres for the shoulder movements. The representation of movements of the hallux, or great toe, has been determined to be about the junction of the middle and posterior thirds of the leg area, and that of toe movements most strikingly at its posterior part. Some investigators place the centres of representation for movements of the toes almost as far back as the parieto-occipital fissure. The ankle representation is probably situated between that of the knee and the areas for the great and small toes. On the mesal surface of the hemisphere in the general area for the lower extremity the centres of representation of movements of flexion at the knee and toes, of extension at the hip, and also of some other movements of the foot and the small toes, have been determined as shown in Fig. 231.

FIG. 231.



Mesal aspect of the human cerebrum, showing areas of representation for movements.

Representation of Trunk and Spine Movements.—So far as has yet been determined by experiment and clinicopathological observation, spinal and trunkal movements are most strongly represented on the mesal aspect of the hemisphere, cephalad of the area for the lower extremity. This representation seems to extend a

short distance over to the lateral surface, as indicated in Fig. 228. When the electrodes were applied to the lateral surface of the hemisphere near its edge, and most strikingly when applied to the mesal aspect, Horsley and Schäfer obtained arching and rotation of the lower spine and pelvis. One or two cases have been recorded in which trunkal spasm or paralysis was apparently due to cortical lesions.

Subareas for the Upper Extremity.—In Fig. 228 the arm or upper extremity area is seen in the mid-Rolandic region, occupying a larger space than either of the other great subdivisions of the motor zone. Numerous experiments and observations have shown that this area can be subdivided in an almost regular manner from below upward for the representation of movements of the chief segments of the upper limb in the same direction, that is, for the thumb and fingers, wrist, elbow, and shoulder. At the upper confines of the general area the representation of shoulder movements blends with that for movements of the lower extremity, and especially for those of the hip and knee; while at the lower boundary the representation of thumb and finger movements blends with that for upper face movements. Excitation experiments at these positions call forth movements of both upper and lower extremities in the one case, and of the upper extremity and face in the other. It will not be necessary to recall the numerous experiments and reported cases indicating the subdivisions of this zone. One important fact determined is that the representation of the upper extremity is much greater cephalad than caudad of the central fissure. The positions of the foci of representation of nearly all the small segments of the upper limb have been gradually differentiated.

Subareas for Face Movements.—Somewhat closely crowded together around the foot of the central fissure are the centres of representation of the various important movements of the face, as determined by many experiments and many reported cases. The general results of the observations and investigations may be summarized in the statement that, as shown in the figure, the movements represented in this area are, from above downward in order, for the orbiculopalpebral movements, for movements of the angle of the mouth, and for movements of the lips and tongue. The platysmal movements are probably represented in the posterior and inferior portion of this general area.

Subarea for Movements of the Lower Jaw.—Horsley has placed the centre for masticatory movements, that is, for the movements of the lower jaw, just behind the laryngeal and pharyngeal centres, near the lips and tongue representation. During an operation he found that electrical stimulation of the precentral convolution at about the junction of the upper and the middle face area caused lateral movements of the jaw and of the angle of the mouth.

Laryngeal and Pharyngeal Representation.—Now and then is seen a case of hemiplegia due to cerebral lesion in which aphonia or some change of intonation is present. These cases in my own experience have generally been left monoplegias, which would seem to bear out the view of Seguin that laryngeal representation is better differentiated on the right than on the left side of the cerebrum. A case of Seguin, and the experiments of Krause, Semon and Horsley, Ferrier, and Masini, indicate the probable location of this centre near the precentral fissure, cephalad of the areas for the pharynx, lower jaw, lips, and tongue. Bilateral extirpation or bilateral lesions seem to be necessary in some instances to produce decided phonatory impairment. Garel has reported a case of cortical vocal paralysis, with the details of an autopsy and a sketch of the locality of the lesion. The inferior portion of the precentral gyre on the *right* side was slightly adherent to the meninges. The membranes being stripped, the surface beneath presented a light yellow discoloration. At the foot of the third frontal gyre were two points of red softening involving almost solely the cortical substance at the upper part, very slightly invading the white. The lesion of the precentral also penetrated slightly into the white substance. The most important of Horsley and Semon's conclusions were (1) that in the monkey is a small area at the lower and anterior portion of the foot of the precentral gyrus, excitation of which produces complete adduction of the vocal cords (bilateral action); (2) that around this area is also represented abduction of the vocal cords, but only feebly and in association with other movements—as those of deglutition; (3) that unilateral extirpation of the whole region produced no appreciable paralysis of the glottis closers or openers. From a general study of the subject of laryngeal cortical representation, Delavan arrives at conclusions as follows: (1) that unilateral irritation of a given cortical centre excites the corresponding bulbar centre and causes bilateral movement; (2) that unilateral destruction of a given cortical centre is without result, as the influence of the opposite cortical centre is sufficient to excite the corresponding bulbar centre and thus to cause bilateral movement; and (3) that bilateral destruction of a given cortical centre causes paralysis. A few experiments apparently show that pharyngeal movements are represented in regions of the cortex closely related to the laryngeal centres, probably a little more caudad. Laryngeal, pharyngeal, and masticatory movements have an almost equal bilateral cortical representation, the cortical centres for each side being sufficient for the movements of both sides.

Representation of the Movements of the Head and Eyes.—As shown in Fig. 228, various movements of the head and eyes are represented in a large area of the cortex,—the most cephalic of the great subdivisions of the motor zone. This area on the lateral aspect

of the hemicerebrum occupies the posterior portions of the superfrontal and medifrontal convolutions, and the upper part of the posterior portion of the subfrontal convolutions. Ferrier, Beevor and Horsley, and Mott have made valuable contributions to our knowledge of this cortical region. Ferrier found that irritation of the posterior extremities of the superfrontal and medifrontal convolutions caused deviation of the head and eyes to the opposite side. Destruction of these centres, according to some authorities, causes conjugate deviation to the side of the lesion. Beevor and Horsley studied the separate representation of the movements of the head and those of the eyes, and also of their associated movements. The movements of the head studied were simple lateral deviation with elevation of the muzzle (in dogs), and adduction with rotation of the head to the opposite shoulder. The movements of the eyes studied were simple opening of both eyes, turning of the eyes to the opposite side and upward, the same deviation with downward inclination, and partial or complete return of both eyes to the middle line; also movements of contraction and dilatation of the pupils. They found that movements of the head had a larger cortical representation than those of the eyes alone. Mott succeeded in making three subdivisions of this area for the movements of the head and eyes,—one for lateral deviation and downward inclination in the superfrontal convolution near the edge of the hemisphere and extending over into the marginal lobule, a second for simple lateral deviation, farther removed from the longitudinal fissure, and a third for lateral deviation or rotation to the opposite side with upward inclination, still lower down towards the Sylvian fissure. The position of these subareas is indicated in the diagram; but it must be borne in mind that experiments failed to demarcate them sharply in every instance. Most of the special movements of the eyes, like those of opening and shutting the eyelids, turning the eyes to one side, and dilatation of the pupils, have their representation near the horizontal limb of the precentral fissure. In a case of my own, during an operation, the careful application of a weak faradic current to a spot in the posterior part of the medifrontal convolution caused distinct deviation of the head to the opposite side. Lesions of a destructive character affecting this region do not often cause persistent paralytic deviation of the head and eyes. Ferrier, Grasset, Landouzy, Wernicke, and Henschen, among others, have seen conjugate deviation produced by irritations of the cortex of the inferior parietal lobule, and some of these observers have noted the same effect from irritation of the supertemporal convolution; but movements of this kind are probably secondary to the visual and auditory sensations evoked. Persistent conjugate deviation is most likely to be dependent upon lesions of the pons and oblongata, because of destruction or irritation of oculomotor nuclei or root fibres.

A special centre for the movements of elevation of the upper eyelid is probably situated in the posterior portion of the medifrontal convolution, just above the face area, and close to the areas of representation for upward and lateral deviation of the head and eyes. In rare instances unilateral ptosis has been found associated with hemiplegia, the cortical lesions being at the base of the second frontal gyre. In a few other cases the same symptom has been present with lesions in the vicinity of the angular gyre, but the explanation of this is probably similar to that which accounts for lateral rotation and other movements of the head and eyes from stimulation of this portion of the visual region: because of the destruction of this important portion of the visual cortex, the eyes, and with them the lids, fail to respond in the usual manner to visual excitations. Mott found that the result obtained by bilateral stimulation of identical points in the areas for lateral deviation of the head and eyes was invariably, if the eyes were not already looking straight forward, to bring them into this position and to produce visual fixation upon a distant object. Other experiments of bilateral stimulation of the areas of representation for upward and downward inclination gave results of a character which in a similar way corroborated those produced by unilateral stimulation. By stimulation of the occipital visual area he produced results similar to those obtained by frontal excitation; but a very weak stimulation of the frontal area sufficed to overcome a strong occipital excitation. Other results of great interest were obtained by this experimenter.

Unilateral and Bilateral Representation in the Cortex.—One of the most striking facts brought to light by the study of cortical localization is that the centres of representation for specialized movements are highly differentiated on both sides of the cerebrum. Another fact, of equal importance, is that the centres for such highly evolved functions or faculties as word seeing, word hearing, speech, and writing are especially developed in the left hemicerebrum, although they have some representation in both hemispheres, and one side of the brain can probably be gradually educated to substitute the other. Those movements of the two sides of the body which are always or usually consentaneous have what is termed a cortical bilateral representation; the centre in either hemisphere can act for movements of both sides. The bulbospinal centres related to these movements are almost equally innervated from each hemisphere, the commissures of these centres conveying impressions to and fro with great freedom. Destruction of the cerebral centre of one hemisphere does not cause paralysis of those muscles which are completely bilateral in their actions, as, for instance, the muscles concerned in respiration and phonation, and the trunkal muscles in general. Paralysis of those movements which are usually bilateral but which have some unilateral differentiation, while it may be recog-

nizable, is commonly less marked than that which occurs in parts of the body possessing highly specialized functions. The movements of the orbicularis palpebrarum or of the masticatory muscles may be impaired to only a slight degree by lesions which destroy their centres, while paralysis of the arm or leg from a lesion proportionately no more destructive may be much more complete and persistent. It is because of this bilateral representation that we have comparatively few records, with autopsies, of laryngeal, pharyngeal, and masticatory paralysis or spasm from cortical disease. In the ambidextrous, and in those who are altogether left handed but have acquired fair use of their right hands, cortical representation is probably bilateral.

Border Centres and Overlapping Areas.—According to Ferrier, the different centres of representation of specialized movements are separated from each other by hard and fast lines; according to others, they merge into each other or overlap; but all authorities seem to agree that the cortical representation of every movement, whether it be of a large or of a small segment of the body, is most concentrated at some one spot. Electrical irritation of an area extending considerably beyond such a point may, however, call forth the movement in question. Horsley holds to the existence of true border centres, irritation of which may elicit movements of different segments of the body at the same time in almost equal degree. Movements of the face and of the upper extremity, and particularly of the thumb, fingers, and wrist, may be evoked by stimulation of the cortex at a position corresponding to the boundary between the arm and face areas as given in Fig. 228. Such localities may be regarded for convenience as border centres. Because of the real or apparent merging of representation, difficulties sometimes arise in fixing the primary focus of a lesion. Two signal or initial symptoms may apparently be present, as when it is difficult or impossible to determine whether an attack of spasm begins in the face or in some portion of the upper extremities.

The Representation of Cutaneous and Muscular Sensations.—In the diagrams Figs. 228 and 229 the posteroparietal convolutions, precuneus, and gyrus fornicatus are designated as sensory areas, by which is meant that they constitute a region of representation for those forms of cutaneous and muscular sensibility which are conducted directly or indirectly to the cortex. The excitations conveyed are those which give rise to touch, pain, and temperature impressions, and modifications of these, such as sensations of pressure, location, and the other sensations which enter into the so-called muscular sense. It is most likely that the fillet radiations pass in part to the thalamus and thence to the cortex, and in part directly to the cortex of these convolutions, expanding as they proceed, their extensions, as already shown, probably ramifying in the molec-

ular layer of the cortex, there to come into contact with the apical processes of the great pyramidal and ambiguous cells of the Rolandic and other regions of the cerebrum. A destructive lesion of sufficient depth to involve the entire cortex of this region will give sensory symptoms, such as various types of anesthesia, and hyperesthesia should result from irritative lesions. The facts of experiment and of clinicopathology are in accordance with this hypothesis. Abundant records show that hemianesthesia results from lesions of the hinder third of the posterior limb of the internal capsule and adjoining portions of the corona radiata. Flechsig, Bechterew, and others have traced the fillet radiations into the posteroparietal regions. Ferrier found that lesions of the hippocampal and adjoining regions diminished or abolished tactile sensibility on the opposite side of the body. Horsley and Schäfer found that partial or complete hemianesthesia resulted from experimental lesions in the limbic lobe, particularly in the gyrus fornicatus, the loss of sensation sometimes being limited to a particular portion of the limbs and trunk. Savill has reported two cases in which anesthesia followed lesions of the gyrus fornicatus; Sharkey has published a similar case; and numerous cases have been reported in which loss or impairment of muscular or cutaneous sensibility has been present with lesions of the posteroparietal convolutions. On the other hand, innumerable cases have been reported of lesions of the motor cortex without the slightest impairment of sensibility. In several cases of excision of the human cortex in the Rolandic region by surgical operations, careful studies of the patients, by the writer and others, failed to show any impairment of sensation. Opposed to these cases, however, are those which have been collected by Dana, Starr, and other observers, which have shown more or less limited losses of sensation from lesions near the central fissure. Ransom and Dana have recorded cases in which irritation of the human motor cortex during operation caused subjective anesthesia of the parts represented in the region of the cortex upon which the experiments were made. The explanation of these cases and of similar experimental facts, such as have been recorded by Mott and others, is to be found in the statements already made with reference to the extensions of the fillet radiations to the cell processes of the motor region. It is evident that sensory excitations which call out motor activities must be transferred from the fillet radiations to cells and processes which convey impulses to the bulbospinal centres and muscles. Destruction of these cortical sensory termini, or of the cells and processes which constitute the field of conjunction which associates the fillet radiations with the processes of the great pyramidal and ambiguous cells in the Rolandic region, may give rise to temporary subjective and perhaps even to objective sensory disturbances. On the supposition that the sensory and motor areas of

the cortex are distinct, Charcot has suggested that sensory phenomena are not superimposed upon motor, but that the former are transient, and that the phenomena, chiefly anesthesias, are functional, and in most respects similar to those which are present in hysterical cases. Andriezen speaks of the cells whose processes in Exner's plexus of the molecular layer receive the terminals and collaterals of the fillet radiations, as the first sensory cells of the cortex. These include the great pyramidal cells, which have been hitherto almost universally regarded as motor. If we are to retain the terms sensory and motor cells, it would seem more reasonable still to adhere to the designation motor for those cells which lie in the region of the cortex in which the first steps in the so-called voluntary motor processes begin. Destruction of the deep layer of the cortex in the Rolandic region, which must involve destruction of these cells, always gives rise to impairment or abolition of movement, and in only a comparatively small number of instances to affections of sensation. Starr and McCosh have reported a case which seems to give conclusive proof that the muscular sense has a localization of its own entirely independent of the motor impulse. The case was, the reporters say, comparable to an accurate physiological experiment upon the cortex of the brain, although no such experiment was intended. It was one of traumatic epilepsy characterized by psychical attacks and headache. Trephining and the removal of a small angioma were followed by loss of muscular sense in the right arm lasting six weeks. The operation was performed upon a spot in the brain about the junction of the superior and inferior parietal convolutions, clearly posterior to the post-central convolution, and resulted in a loss of muscular sense in the opposite hand and forearm, without any disturbance of other sensations or of movement.

Visual Localization.—The occipital lobe, and the adjoining parietotemporal area, usually spoken of as the angular convolution, together constitute the cortical organ of vision as indicated in the diagrams Figs. 228 and 229. A distinction is sometimes made between *primary cortical* and *secondary cortical* visual centres, the primary cortical centres, according to this view, being situated in the cuneus and about the calcarine fissure, and the secondary cortical centres, which are those for higher visual perceptions, being in the angular gyre and anterior portion of the occipital lobe. Even in the higher visual processes excitations are probably first received in the primary cortical visual areas of both hemispheres, to be carried thence to the higher visual centres of each side. Confusion, however, must be avoided in the use of these terms when speaking of visual centres. The term primary centre for vision is more commonly applied to the basal terminations of the short or central reflex optic path, that is, to the pregeminum and pregeniculum, and,

according to some, to the pulvinar of the thalamus. According to this view, the cortical areas just described are secondary visual centres. It is best to speak of the lower centres as basal or reflex, and of the others as cortical or higher centres, recognizing the fact that in cortical vision we have primary and secondary processes.

Subdivisions of Retinal Representation.—Particular regions of the retina probably bear more or less definite relations to particular portions of the cortical visual area, as advocated especially by Munk; but the only localizations so far definitely determined would seem to be those for correlated halves or segments of the retina in the region of the calcarine fissure, and for macular vision in the angular gyre and anterior portion of the lateral aspect of the occipital lobe. Cortical visual representation has been the subject of much investigation and discussion by physiologists, as by Munk, Ferrier, Yeo, Monakow, Ganser, Blaschko, McKendrick, Horsley and Schäfer, Luciani and Tamburini, Dalton, Bianchi, Hitzig, Goltz and Loeb. Cases of hemianopsia with autopsies have been recorded by Jastrowitz, Haab, Hun, Féré, Sharkey, and many others; and valuable compilations of these cases have been made especially by Seguin and by Henschen. Von Gudden and his followers have applied the atrophy methods to the same study, with results in the main confirming the views of the physiologists and clinicopathologists. When the subjects of hemianopsia and other visual defects and disturbances are discussed in detail, some of the questions connected with cortical visual representation will receive further elucidation. It is only necessary to remember here that hemianopsia is blindness of half the visual field, and that in its most usual form this blindness is bilateral and homonymous, that is, of correlated halves of both eyes, while the term amblyopia is applied to general loss or diminution of vision. Experimental and pathological observations are conclusive that a unilateral lesion on the mesal surface of the hemisphere in and around the calcarine fissure is capable, according to its size, and probably according to its exact position, of producing either lateral homonymous hemianopsia or a lateral quadrant or sector defect of vision. Lesions of the calcarine cortex of both hemispheres cause double hemianopsia more or less complete according to their extent. Lesions of this visual area rarely give perfect hemianopsia, probably because disease usually causes irregular cortical destruction. According to Henschen, the elements of both retinal halves are represented by different groups of cells, which, however, lie side by side. After total loss of vision in both eyes, complete atrophy of these cortical visual cells has been found; but after loss of sight in only one eye, normal cells have been found in considerable number alongside of others which were granular and pigmented.

Macular Representation.—Ferrier found that unilateral lesion

of the angular gyre produced amblyopia of the opposite eye, but that the defect of vision was not of the hemiopic variety; and he also determined that bilateral destruction of the same cortical areas produced more or less permanent impairment of visual acuity on both sides. These and other experiments and various clinicopathological and surgical observations indicate that in each angular region and in the anterior portion of the lateral occipital lobe is located the representation of macular or central vision, most markedly for the opposite eye, but in some degree also for the eye of the same side. It is also the area for the visual memories of language, that marked word vision in Fig. 228. When this region is not involved in experimental or pathological lesions of the occipital lobe which produce hemianopsia, a small area of clear vision remains, as has been demonstrated particularly by a number of cases of double hemianopsia reported by Förster, Schweigger, Groenouw, Gaffron, Schmidt-Rimpler, Magnus, Dunn, and others, in which, with more or less complete double hemianopsia, central vision was either retained or after having been lost was subsequently restored in whole or in part. Such cases of double hemianopsia sometimes lose the sense of locality, becoming unable to orient themselves. Dunn's patient could not form a conception of the geography of his own house nor of any place where he had ever been, and Dunn has therefore suggested the existence in the brain of a centre, which he would name for convenience the *geographical centre*, where are recorded the optical images of locality.

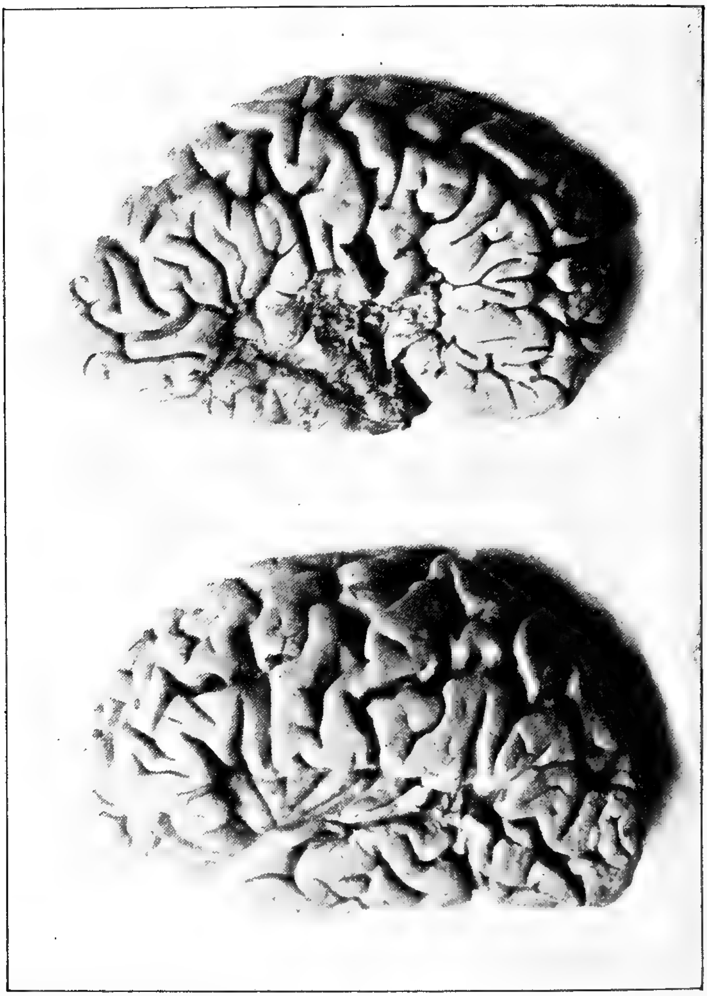
Auditory Localization.—The cortical localization of hearing in the first and second temporal convolutions is as well established as that of any cerebral function. Since Ferrier's early localization of audition in this region, experiment and observation have been in general accord in confirming his determination, although good authorities have claimed a larger area in the temporal lobe for the function of hearing. The cortical area for the reception of auditory impressions, like that for vision, is doubtless a compound one. It is probable that a large district of the upper part of the temporal lobe is concerned in a general way with audition, and might be designated as the auditory sphere or the primary auditory cortical zone; while in a limited portion of this general region is situated an area in which the perception of words takes place, or which is the centre for the auditory memories of spoken language. In other words, we have primary and secondary areas for hearing in the cortex, just as we have lower and higher cortical visual centres. Destruction of almost any portion of the supertemporal convolution, and particularly of its middle and posterior parts, will cause interference with hearing, usually with reference to the ear of the opposite side. When, as a result of experiment or disease, a portion of this convolution has been destroyed, the hearing, lost for a time in

one ear, has subsequently been regained, showing the possibility of a complete or almost complete compensation by the convolutions on the opposite side, or an assumption of the auditory function by adjoining portions of the convolution affected. In order that complete cerebral deafness should result, it is necessary that a destructive lesion should involve the supertemporal convolutions of both sides. The limited area for word hearing, lesion of which causes the symptom known as word deafness, is situated, as shown in Fig. 228, in the posterior parts of the first and the adjoining superior portion of the second temporal convolution. Cases demonstrating both the general and the special localizations just noted have been recorded by Bastian, Wernicke and Friedländer, Shaw, and the writer. My own case seemed to include all the requirements of a complete experiment, the cerebral lesions having been bilateral, as shown in Fig. 232. At the posterior extremities of the supertemporal and meditemporal convolutions was a depression evidently the remains of an old embolic cyst, the supertemporal convolution of the same side being remarkably attenuated in its posterior two thirds. On the right side, the supertemporal gyre, a large portion of the meditemporal gyre, the insula, the retroinsular convolutions, the lower extremities of the central convolutions, and a large extent of the ganglia and capsules were destroyed by an old hemorrhagic cyst. This patient had suffered from two apoplectic attacks, the first leaving her word deaf, total deafness occurring after the second attack. In various other ways confirmation of the localization of hearing in the upper temporal convolutions has been afforded, as through cases in which auditory discharges have been present with irritative lesions of these convolutions.

The Naming Centre.—A disputed localization is that which has been variously termed the *naming centre*, *idea centre*, or *concept centre.* The conception of an object obtained through the different senses is symbolized by name, and Broadbent suggested the existence on the sensory side of the nervous system of a structural arrangement for this process, which he proposed to call the *naming centre;* to it converge the tracts from the receptive regions of the cortex, and from it pass tracts which go to the motor and executive centres. Cases of verbal amnesia, and especially cases in which the patient has lost the memory of names or nouns which symbolize objects, are frequently observed, and in some instances at least this symptom is due to destruction of this naming region, or of the fibres which enter or leave it. The evidence is, on the whole, in favor of the setting apart of such an area. A case seen by me in consultation with Dr. Wilson Bowers and Dr. J. W. McConnell indicated the situation of this centre, the localization being in confirmation of a speculation of Broadbent. This patient was word blind in large part, but was not word deaf. She could not name objects either

from sight or from touch, although she evidently knew what they were. She talked spontaneously, but with difficulty, and did not use concrete nouns. On autopsy a tumor was found involving the

Fig. 232.

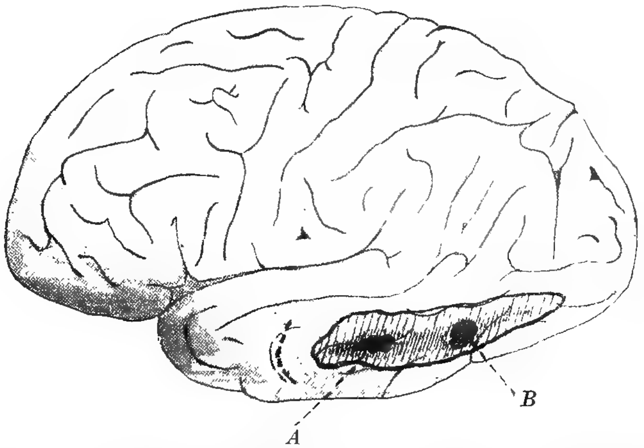


Lesions of the superior temporal convolutions of both hemicerebrums: the upper figure shows a hemorrhagic cyst of the right hemisphere destroying the two upper temporal convolutions and other parts; the lower figure shows in the left hemisphere a cyst or cavity the result of an old embolism, and also the atrophied supertemporal continuous with the retroinsular convolution The patient was word deaf and eventually totally deaf.

under surface of the temporal lobe. (Fig. 233.) The surface of the third temporal convolution in its posterior half, and to a much less extent of the second and fourth temporal convolutions, presented a slightly disintegrated appearance. Internally the parts chiefly destroyed were the white matter of the third, to a less ex-

tent of the second, and to a still less extent of the fourth temporal convolution. The disease almost certainly started in the third temporal convolution, in which I would locate this centre.

FIG. 233.



Tumor of the meditemporal convolution, indicating the position of the naming centre: *A*, densest and probably oldest portion of the growth; *B*, place where a nodular mass was torn away during the autopsy; the heavier shading indicates the region of the greatest cortical and subcortical destruction. The dotted line shows the anterior subcortical limit of the lesion.

Olfactory Localization.—The exact limitations of the cortical regions concerned with the sense of smell have not yet been determined. According to Broca and Zuckerkandl, the entire limbic lobe takes part in olfactory sensation. According to Andriezen, the cortical regions which receive the olfactory projection fibres are (1) the genual or precallosal part of the gyrus fornicatus; (2) the septum lucidum; and (3) the inferior extremities of the uncinate and hippocampal gyres. Certainly the first and last of these regions when diseased or injured will give disorders of smell. Probably the amygdala and dentate fascia also take part in this function, and it may be that while all the areas mentioned have an important rôle to play in animals in which the organ of smell is highly developed, in man the area is more restricted. Hill's views with reference to the functions of the fornix would seem to be in accord with this idea, this structure having terminal relations with most of the parts mentioned. He looks upon the fornix as containing the continuation of the olfactory tract, chiefly on the grounds (1) that there is a great similarity of structure between the olfactory bulb and the sheath of gray matter (fascia dentata) which invests the margin of the cortex where it is folded over the hippocampus; (2) that the fascia dentata is continued up beneath the fimbria for a distance apparently varying directly with the animal's acuteness of the sense of smell; (3) that the fornix is of much smaller cross section in the nonsmelling

marine mammals than in animals with a moderate sense of smell; (4) that there is reason to think that in mammals the cerebral hemisphere twists over upon itself during its growth in such a way that the olfactory tract would necessarily be folded like the fornix around the peduncles; (5) that in reptiles, and other low vertebrates in which this rotation has not occurred, the olfactory tract is connected with the gray matter lining the third ventricle, which seems to be homologous with the front of the thalamus; and (6) that unless the olfactory tract has such a connection with the central gray tube in the albicantia, the first nerve differs from all other nerves in being immediately connected with the cerebral cortex. Ferrier found that electrical irritation of the uncinate gyre gave indications of subjective olfactory sensations, and cases have been reported by Ogle, Fletcher, Ransom, Hughlings Jackson, McLane Hamilton, Worcester, and others, in which either impairment or loss of smell or olfactory auræ were associated with lesions of the gyrus uncinatus or its immediate vicinity.

Gustatory Localization.—The cortical area for the sense of taste has not yet been positively determined, although the indications favor its localization close to the area for smell, probably in part at least in the fourth temporal convolution. Ferrier, Gad, and Schtscherback have experimented with the view of determining its position. Ferrier's experiments indicated that both smell and taste were affected by lesions of the uncinate convolution or hippocampal lobule and its neighborhood. It is difficult to determine anything positive about the function of taste by experiments on the lower animals, because many acts, as biting, swallowing, and chewing, which have some relation to the function of taste, are of a reflex character. The formation and backward movements of the bolus are probably governed by the cortex. The determinations of Gad and Schtscherback were based upon a study of these movements. The latter's experiments would seem to indicate that the gustatory centre has a much larger extent in the rabbit than that given by Ferrier. He describes it as extending two or three millimetres anterior and posterior to a line corresponding to the coronal suture and to the posterior border of the chiasm upon the entire convex surface of the hemisphere.

Speech Centres.—The general subject of aphasia and cerebral disturbances of speech will be considered in a special section, but in order to complete the survey of cortical localization it will be necessary to refer here to certain speech centres or areas situated on the motor side of the cerebrum. These are (1) the area in the diagram, Fig. 228, marked "speech," which occupies the hinder part of the left third frontal (subfrontal) convolution; and (2) the area designated "graphic," in the posterior part of the second frontal (medifrontal) convolution. The localization by Broca, in 1861, of

articulate language in this part of the cortex, often called Broca's convolution, was the foundation stone of modern cerebral localization. For a time all cerebral speech disturbances were indiscriminately referred to lesions of this convolution, but the cerebral mechanism of speech has been largely worked out in recent years, and disorders of language of cerebral origin are now known to result from lesions variously situated. A study of this mechanism includes a consideration of the various sensory cortical areas already described, and especially of the auditory and visual zones, also of the naming or concept centres, intermediate between the sensory and motor spheres, and of the tracts which connect the receptive, conceptive, and executive centres, also the incoming auditory and visual tracts, and those which carry outward and downward the speech impulses to the bulbospinal nuclei. It follows that numerous forms of cortical and subcortical aphasia are now recognized. The form which is dependent upon lesion of the left third frontal convolution is usually spoken of as *motor aphasia* or *aphemia*. Total destruction of this region in right handed adults makes speech impossible, at least temporarily. Broadbent has described this region as a propositionizing centre, as that part of the cortex in which thoughts are set into a framework of words, the utterance of these words being consummated through centres situated at the bases of the central convolutions. Such a subdivision into *propositionizing* and *utterance* centres has a practical value, as a lesion of each of these regions has its own symptomatology. In the utterance centre or region are located the representations of all parts concerned in articulation, as of the larynx, pharynx, tongue, lips, and face. The subcortical speech tract probably proceeds from this region, although some hold it to be distinct. According to some authorities, Broca's convolution is the storehouse for the guiding psychomotor images or memories of words, these being regarded as largely sensory, that is, made up of memories of tactile and muscular sensations. Wyllie speaks of Broadbent's utterance centre as the region containing the motor executive cells for speech, the place where speech becomes locally exteriorized. Lesion of this area gives rise to an articulative paresis or paralysis, not to a disorder of speech in a strict sense, such as results from a lesion of Broca's convolution.

The Graphic Centre.—Following good authority, a graphic centre or motor centre for writing has been placed at the caudal extremity of the second frontal convolution cephalad of the area for the lower portion of the upper extremity. It is probable that a pure motor agraphia may result from a lesion in this locality, at least so far as writing with the right hand is concerned, although agraphia usually accompanies destructive lesions of the left third frontal gyre. A few facts have been recorded which seem to indicate that it is possible to have a motor agraphia without either

aphasia or paralysis of the upper extremity. In studying cases of apparent motor agraphia, it must not be forgotten that this defect may be due to word blindness; but the latter can usually be differentiated by the fact that the patients often retain the power of signing their names or of writing simple words through the sense of touch. Broca's centre and the graphic centre are separately indicated on Fig. 228.

The Insula and Retroinsular Convolutions.—The well known topographical relations of the insula with the third frontal, the central, and the inferior parietal convolutions on the one hand, and with the temporal lobe on the other, and also internally with the lenticula, indicate its probable functions. It is altogether likely that its cell and fibre systems are concerned with the functional association of these parts of the brain. According to Cunningham, a study of its developmental progress shows that a part of the insula is connected with the falciform lobe, which may indicate that it also unites this lobe with the frontoparietal cortex. The covering of the insula in man, in the process of development of the convolutions of the lateral surface of the hemisphere, does not indicate that it is a retrogression in the human species. The insula in man is larger and more complicated than in the monkey, in which it is more or less uncovered. In the orang, the gorilla, and the chimpanzee it has three or four gyres or folds, and in man five. The retroinsular convolutions are apparently continuous with the posterior portion of the supertemporal convolution. A close correspondence exists between the fissures and gyres of the insula and those of the lateral surface of the hemisphere, according to Cunningham and Eberstaller, its three fissures being probably comparable with the precentral, central, and retrocentral fissures. Variations in the insula accompany variations of the frontoparietal region. The first temporal convolution sometimes shows transfissuration, as in specimens described by me. The tendency is to confluence between the anterior portion of the supertemporal fissure and the fissure which separates the first two retroinsular convolutions. In my case of cerebral deafness referred to under auditory localization (page 345), the posterior retroinsular convolution, like the posterior portion of the first temporal convolution, was much attenuated. By some of the older writers the insula has been regarded as one of the centres for speech; but it is probable that the affections of speech in reported cases of absolutely limited lesion occur as the result of injury to the speech tracts which connect the centres for word hearing and concepts with Broca's convolution. In Bateman's work on aphasia, and in other treatises upon this subject, a number of cases have been collected in which atrophy, hemorrhage, softening, or other lesions of the insula were present, but only in rare instances have lesions causing disorder of speech been restricted to this part. These probably have been

instances of conduction aphasia, paraphasia, paralexia, and paramimia.

Latent or Undetermined Cortical Regions.—It is doubtful whether any portion, however small, of the cerebral cortex is absolutely destitute of function. Every part of a highly evolved substance, like the cerebral cinerea, must have an important use, but it is nevertheless true that as yet the functions of some areas of the surface of the brain have not been accurately defined, although the so-called latent regions are gradually disappearing with new discoveries. Lesions of the orbital surface of the frontal lobe, of the inferior occipitotemporal convolutions, and of any portion of the right temporal lobe give rise to but few symptoms, although the superior temporal convolutions of this side are probably concerned in audition, but have a much lower differentiation as centres than the corresponding convolutions of the left side. The third temporal convolution has hitherto usually been designated as without known function, but it will be seen from the discussion of the naming centre that it is probable that a form of higher verbal representation is here located.

Different Classes of Localizing Symptoms.—The cortical areas of representation just described must be made use of after a definite plan, if a knowledge of them is to be turned to the best account. At least six classes of symptoms may need to be considered, namely, those of (1) local irritation; (2) local destruction; (3) local pressure; (4) invasion from adjacent areas; (5) local instability; and (6) reflex action at a distance. In the motor zone the especial symptom of irritation is spasm; but irritation symptoms may occur in other localities, as in the visual, aural, olfactory, gustatory, or cutaneous areas, where they may take the form of hallucinations or other perversions of the senses. Symptoms indicating destruction are, in the motor areas, paresis or paralysis, and, in other regions, such manifestations as hemianopsia, word or mind blindness, word deafness, anesthesia, analgesia, anosmia, and ageusia. Pressure and invasion symptoms may be indicative either of irritation or of destruction, but are considered by the clinician in their relations to areas near the seats of the lesions. Invasion symptoms will at first commonly be phenomena of irritation, and later both of irritation and of destruction. By symptoms of instability are meant those manifestations that occur as the result of discharges of unstable cortical areas, which may or may not be the seats of demonstrable gross lesions. Reflex symptoms will occur mostly in connection with lesions of the cranial or other nerves, and of the cerebral membranes, particularly of the dura. They are sometimes sources of error, especially in motor localization. A study of the initial symptom or sign in a case of irritative cerebral lesion, and also of the serial order of phenomena, may be of great importance. Seguin has

proposed to call this initial symptom the "signal symptom." In every part of the motor cortex which can be examined experimentally there is represented a definite movement or combination of movements, being the primary movement which is elicited by minimal stimulation only; the secondary movements are due to the subsequent invasion by the discharge of nerve energy of those portions of the cortex which lie nearest to the parts stimulated and with which they are in close relation. (Horsley.) The primary movement gives the signal symptom of Seguin, and the secondary movements represent the "serial order" of phenomena.

FUNCTIONS AND LESIONS OF THE BASAL GANGLIA, CAPSULES, AND CENTRUM OVALE.

Functions and Lesions of the Striatum (Caudatum and Lenticula).—The striate bodies in the embryo, as shown by Flechsig, Wernicke, Edinger, and others, are related to the cortex. Even in the adult human brain, as shown in several illustrations in this work, they are seen to have direct although slight cortical connections. According to Ferrier, in man and in the monkey little difference exists between the results of complete destruction of the cortical motor centres and of that of the striatocapsular region; but lesions of the striata, if the internal capsules are not directly or indirectly affected, do not cause permanent impairment of motion or sensation. He concludes that both parts of the striatum are centres of innervation of the movements differentiated in the cortex, but that they are of lower grade of specialization. Ziehen found that their stimulation gave rise to contractions similar to those produced by stimulation of the anterior portion of the motor cortex. Marchi believes that these ganglia have mixed functions of motion and sensation. Danilewsky found that irritation of both the caudatum and the lenticula caused increase in blood pressure, but admitted that this might be due to the diffusion of the current to the peduncles. The experiments of Hale White indicate that the striate body as well as the thalamus, in rabbits at least, has the power of modifying the temperature of the body. Bourneville relates a case of a large patch of red softening in the centrum ovale and a recent patch in the right striatum in which the temperature before death was 104.8° F. White reports the case of a man paralyzed on the right side, who never spoke, nor took any notice of things around him, and who after a month had elapsed had a temperature of 102.4° F., with a fit at the same time. The autopsy showed just above the precommissure a brownish patch about a quarter of an inch in diameter so soft that it left a hole when the brain was cut. Dr. Bagojawlensky has reported a case of elevated temperature where the only lesions were echinococcus cysts in the striatum. (Ott.) In cerebral hemorrhage and softening from embolism the striatum is often involved with the internal

capsule, monoplegia, hemiplegia, and hemianesthesia being present, but when these affections are permanent they are probably always due to capsular lesions. Hemorrhage limited to either of the striate bodies has been known to cause temporary paralysis, but presumably through pressure on the internal capsule. Gowers records a case in which a narrow vertical band of softening extended through the length of the lenticula, no paralysis having been present, and even a recent hemorrhage limited to this ganglion has caused no symptoms. Cases of athetoid and choreoid spasm have been observed in connection with lesions of the posterior portion of the lenticula, but either the internal capsule or the thalamus or both have usually been found implicated. Future investigations may give facts which can be turned into the channels of diagnosis, but the striatum at present may for clinical purposes be regarded as an uncertain region.

Pseudobulbar Paralysis from Lesions of the Lenticula.—In some of the cases of so-called pseudobulbar paralysis in which patients suffer from anarthria, or paresis of articulation, similar to that observed in paralysis of bulbar origin, among the most constant lesions found have been those in one or both lenticulas. In one of the earliest reports of such a case, by Kirchoff, a focus of softening surrounded by sclerosed tissue was found in the lenticula, disease of the pons and oblongata being absent. Ross, Magnus, Nothnagel, Drummond, Barlow, Rosenthal, Féré, Jolly, Oulmont, Fuller, Browning, the writer, and others have also recorded cases. Boulay has collected thirty such cases, twenty-four of which were followed by autopsies; in nearly all the cases the lesions, variously situated, were bilateral, and frequently they were symmetrical. Some were in the cortex, particularly affecting the lower extremities of the central and posterior extremities of the second and third frontal convolutions. As a rule, the glossolabiolaryngeal symptoms were not so marked in the cortical as in the subcortical and ganglionic cases. The lesions were in the cortex, lenticula, caudatum, thalamus, capsules, and centrum ovale, but the lenticula, and particularly its outer segment or putamen, was most frequently diseased in the typical cases. The most common lesions were foci of softening and of hemorrhage, the patients usually being aged and their vessels highly atheromatous.

Functions of the Thalamus.—The physiology of the thalamus is still largely unsettled. Between the thalamus and the cortex, and between it and the parts below, run innumerable fibres. According to one view, the thalamus receives from the sensory tract impressions which are transmitted to motor centres in the striatum, the two sets of ganglia forming the two sides of a great reflex arc. Removal of the thalamus, or destruction of that part of it in the neighborhood of the inspiratory centre in the wall of the third ventricle, was found to influence coordinated movements in the rabbit. (Christiani.) Meynert believed that the thalamus was connected in

function with the upper extremities; but the facts of disease and experiment indicate that this ganglion is related to the face and leg as well as to the arm; that it is essentially an organ for one half of the body, as is also shown by its position and its relations with other parts of the brain. According to one view, the thalamus receives sensory impressions before they are transmitted to the cortex; and the experiments of Monakow led him to the conclusion that different portions of the thalamus are related to different cortical areas. Crichton-Browne regarded this ganglion as a great centre of general sensibility. Others have assigned to it motor functions. The weight of evidence, both from experiment and from disease, connects the thalamus with various forms of sensation. One view is that the thalami fulfil the same functions for tactile impressions that the pregeminum does for visual impressions; that is, that these ganglia are the centres of the *spatial senses*, vision and touch, the centres in which the sensations are referred to positions in space. They are centres intermediate between special sensory centres and the higher centres of the cerebrum. (McKendrick.) The functional subdivisions of the thalamus have not, however, as yet been accurately determined. According to the alluring theory of Luys, it can be subdivided from before backward into four ganglionic masses,—an olfactory, a visual, a general sensory, and an auditory centre,—these special cell nests being in relation, by means of definite fasciculi, above with the cortex, and below with the peripheral sense organs. This view of Luys's cannot be regarded as demonstrated, although portions of the thalamus or of its projecting masses have been shown to be related in some way to common sensibility, to sight, and to hearing. Numerous experiments, but especially those of Ott and Hale White, indicate that the thalamus or some portion of it is also a heat-regulating organ. Elevations of temperature resulted from puncture of the thalamus, but Ott has shown that in puncturing the anterior end of the thalamus the tuber cinereum may be wounded, and this structure has been demonstrated to be a centre both of thermotaxis and of polypnea or rapid breathing. Podanowsky and Popoff from a study of antipyretics, with division of the brain behind the striata, conclude that an especial vasomotor centre exists in the anterior part of the brain; and Ott found that puncture of the anterior half of the thalamus nearly always caused blood pressure to fall, and that it never rose again to the original height, although the number of the pulsations remained about the same. Faradic irritation of the thalami, which, however, is not always to be absolutely trusted, because of spreading of the current, causes a rapid rise of blood pressure. Ott concludes that vasotonic centres are situated in the cephalic portion of the thalamus, and that these centres probably exercise some control over the monarchical vasomotor centres in the oblongata.

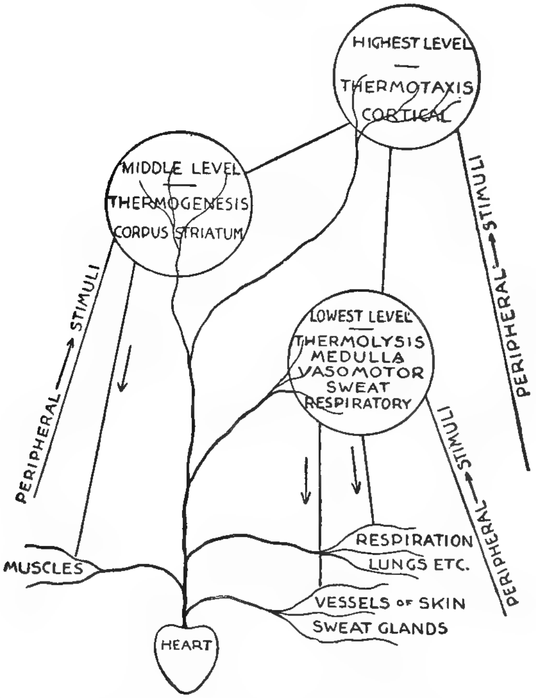
Symptoms in Cases of Thalamic Disease.—In reported cases of disease of the thalamus the lesions have almost always involved other parts. The symptoms have been sensory, motor, visual, auditory, olfactory, thermic, vasomotor, and psychic. All the forms of common sensibility (cutaneous and muscular sensations) have in some instances, as in one case of my own, been impaired or lost. Among motor symptoms, paralysis, ataxia, contractures, forced positions, particularly of the head and upper extremities, spasm, and athetoid and myotonic phenomena, have been observed. Among visual symptoms have been general amblyopia and hemianopsia, but particularly the latter, although I believe this is more commonly due to lesion of the pregeniculum, which can scarcely escape involvement in gross disease of the pulvinar of the thalamus. Impairment of hearing has been noted in very rare instances. Among vasomotor symptoms which have been reported are hemorrhage and vasomotor paresis or spasm. Elevation and depression of temperature, and stupor, mental slowness, and irritability, are also among the recorded phenomena. Most of the cases in which paresis or paralysis has occurred can be explained on the view that they were due to pressure or to the extension of the lesion to the internal capsule. In one case, cited by Debierre from Hunter and A. Voison, the patient lost successively in three years smell, sight, hearing, and general sensibility, and remained insensible to all external sensory excitations; the thalami were found destroyed by a neoplasm.

Heat Centres and the Thermic Mechanism.—Physiologists have experimentally demonstrated the existence of heat centres in various parts of the neuraxis. A thermotaxic centre is one concerned in the regulation of the temperature of the body. A thermogenic centre is one concerned in the production of heat, its irritation causing the evolvement of heat, and its destruction the diminution of heat. Thermolysis is the dissipation of heat, and thermolytic centres are concerned with this process. The following table by Ott presents in a compact form the views of this physiologist, which are based both upon his own experiments and upon a study of the literature of the subject.

FOREBRAIN	*Cortex.*—Thermoinhibitory centres: (1) cruciate; (2) Sylvian. *Base.*—Thermogenic centres: (1) caudate nucleus (caudatum); (2) gray matter of septum lucidum (White); (3) gray matter in front of and beneath caudatum.
INTERBRAIN	Thermogenic centre in the tuber cinereum. Polypneic and vasotonic centres in the tuber cinereum, connected with thermolysis.
AFTERBRAIN	Thermolytic centres.—Respiratory and vasomotor.
SPINAL CORD	Thermolytic centres.—Sudorific centres.—Thermogenic centres.

According to Hale White, a close parallelism exists between the temperature regulating mechanism of the body and the three evolutionary levels of Hughlings Jackson, which is illustrated diagrammatically in Fig. 234. At the lowest level is the thermolytic portion of the mechanism, consisting of the cutaneous vessels with their vasomotor nerves, the sweat glands with their nerves, and the respiratory apparatus also controlled by various nerves. The thermolytic centres are in the oblongata and spinal cord. The second or middle level of the thermic mechanism is the thermal or heat producing, and its thermogenetic function is presided over by the

Fig. 234.



The three thermic mechanisms of the nervous system. (W. Hale White.)

striatum, either in its caudate or in its lenticular division, possibly also, as shown by Ott's table and in the preceding discussion of the thalamus, by centres in the septum lucidum, in front of and beneath the caudatum, in the tuber, and in the thalamus. The third and highest level of the thermic mechanism is in the cortex.

Heat Centres and Clinical Phenomena.—The researches on heat centres and the thermic mechanism have proved of value in the hands of Wood, Ott, Hale White, and others, in explaining the phenomena of fever and the mode of action of antipyretic drugs; but in cases of focal disease of the brain and spinal cord, tempera-

ture phenomena abundantly present have not received the attention which the discoveries in thermic localization should suggest. Interesting and sometimes extraordinary temperature disturbances are observed as the results of such lesions. The temperature may be elevated in one half of the body, or in a limited portion of it, as the result of a hemorrhage or other focal cerebral lesion. According to Bastian, after hemorrhage into one thalamus the temperature of the paralyzed side may for several weeks or months be one or two degrees higher than on the sound side; and the same changes in temperature, but slighter in degree and more temporary, may result from lesions of the striatum. Ott has collected a meagre series of cases bearing on this question of heat centres, to some of which references have been made in preceding paragraphs. He cites two cases which he believes support the view that the cruciate and Sylvian heat centres in the lower animals are represented in the cortex of man. Horsley has reported a case of hyperpyrexia due to punctiform hemorrhages in the pia of the medifrontal convolutions and to a large hemorrhage in the precentral gyrus. The case was one in which he trephined, and the rise of temperature started at the moment of operation and was not preceded by a fall. McAlister has recorded the case of a paraplegic with a remarkably unstable temperature, one day running to 109.2° F. and in three quarters of an hour dropping to 97.8° F., another day reaching 110.4° F. and in half an hour dropping to 98° F. Ott believes that in this case there was a very excitable state of the spinal thermogenic centres, due to impaired restraint from the cortical thermoinhibitory centres and irritation from lesion of the spinal cord. Singular instances of great elevation of temperature in hysterical patients have been recorded, but here the possibility that the temperatures were factitious must be borne in mind. Numerous cases of so-called hysterical fever have been put on record. In order to render more complete the survey of clinical phenomena in their relations to the thermic mechanism, brief reference will here be made to the effects of lesions of the pons on temperature, although its functions and diseases have not yet been discussed. Bastian long ago stated that in apoplexy of the pons, if the life of the patient be prolonged, the temperature on both sides of the body steadily rises, until at the time of death it may have attained 108° or even 110° F. In one of my own cases of pontile softening the rectal temperature was for a time slightly elevated, and shortly before death it rose to 108° F. A few cases of pontile lesion, usually unilateral, are on record in which decided changes of temperature have occurred,—commonly elevation, with differences in the two sides of the body; although in other reported cases the temperature has been below normal. The weight of clinicopathological as well as of physiological evidence is in favor of some change of temperature as the

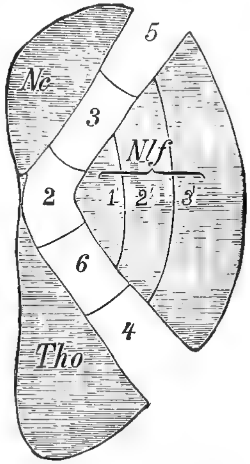
effect of lesions of the pons, thalamus, striatum, tuber cinereum, and of some portions of the frontoparietal cortex.

Lesions of the Geniculate Bodies (Pregeniculum and Postgeniculum).—The tendency of investigators in recent years has been to connect the pregeniculum with the visual tracts and the postgeniculum with the auditory tracts. Von Gudden believed that by his atrophy methods he had demonstrated that the visual centres at the base of the brain were in the pregeminum, pregeniculum, and pulvinar of the thalamus, and also that the postgeniculum and postgeminum had no relations to vision. Forel and many others have practically accorded with Von Gudden in these views. Darkschewitsch doubts the connection of the pulvinar with the optic tract, believing that most of the fibres which go to this body pass through it to the pregeminum. He also casts some doubt upon the relations of the pregeniculum to the optic tract, believing that although fibres of this tract can undoubtedly be traced to it, these may, like those of the pulvinar, pass onward to the pregeminum. One of my cases of thalamic disease, referred to in several places in this work, lends support to this view of Darkschewitsch, two thirds of the thalamus, including most of the pulvinar, having been destroyed without any resulting disorder of vision. Zacher has reported that he has found the postgeniculum attacked by secondary degeneration following destruction of the first and second temporal convolutions. Lekarsky has shown that after softening of the cuneus descending degeneration of the optic radiations, of the pulvinar, of the pregeniculum, and of the capsule of the postgeniculum occurs.

Subdivisions and Lesions of the Internal Capsule.—A knowledge of the position, shape, relations, and functional subdivisions of the internal capsule is of great importance to the clinician, as in a large majority of the cases of hemorrhage and softening from apoplectic attacks more or less destruction of this region takes place. In this narrow strait between the ganglia are concentrated most of the motor and sensory projection fibres, as well as other important fasciculi, and therefore lesions destroying comparatively limited areas may give rise to widespread paralysis and anesthesia. Probably a close study of the symptomatology of cases of monoplegia and hemiplegia, with carefully made autopsies, will enable us eventually to locate with considerable accuracy before death lesions in the internal capsule and centrum ovale as we now localize cortical lesions. Illustrations of varying types of hemiplegia which largely indicate the degree of destruction in the internal capsule will be given in the section on monoplegias and hemiplegias. The general subdivisions of the internal capsule, according to Obersteiner, are shown in Fig. 235, and, from before backward, are a bundle to the thalamus; next the frontopontile tract; about the knee of the capsule, the so-called genual tract, which includes the

fasciculi for the motor cranial nerves; a little posterior to the knee the beginning of the pyramidal tract proper; and still posterior to this, the sensory tract, or *carrefour sensitif*. The anterior segment, probably nearly a third of the anterior half or pregenual half of the internal capsule, occupied by a tract of fibres connected with the thalamus, is sometimes called Meynert's anterior peduncle of the thalamus. The genual subdivision for the motor cranial nerves, according to Debierre, contains from before backward the fasciculi of the inferior facial, the masticatory, and the hypoglossal nerves. The face fibres lie innermost, the arm fibres are in a median position, and the leg fibres are outermost. The differentiation of the internal capsule into its various subdivisions has been gradually carried further and further by various methods, as by pathological observations, by embryological investigations as to the time and manner of appearance of separate fasciculi, by the study of secondary degenerations, and by careful experiments upon lower animals,—experiments by destruction and electrical irritation. The results from all these methods are in general accord. Important experimental studies of the internal capsule have been made by Franck, Beevor and Horsley, and others. The latter suggest that the main axes of the fibres of the internal capsule correspond, so far as anteroposterior arrangement goes, with the cortical foci of representation taken along the lines drawn at right angles to the direction of the upper two thirds of the fissure of Rolando. Placed in order from before backward are eye movements, head and face movements, upper limb movements, and lower limb movements. The arrangement in the capsule, according to them, is but an imitation of that in the cortex, which in its turn is but a peripheric projection of the order of the metameres of the whole body. In Fig. 236 is shown a scheme of this arrangement of the motor fasciculi in the internal capsule. It is supposed by most observers that the optic radiations which come from the pregeminum, pregeniculum, and possibly from the pulvinar, pass through the most caudal portion of the sensory subdivisions of the internal capsule. Obersteiner, Debierre, and others also speak, but with less certainty, of the olfactory radiations passing in this portion of the capsule.

FIG. 235.

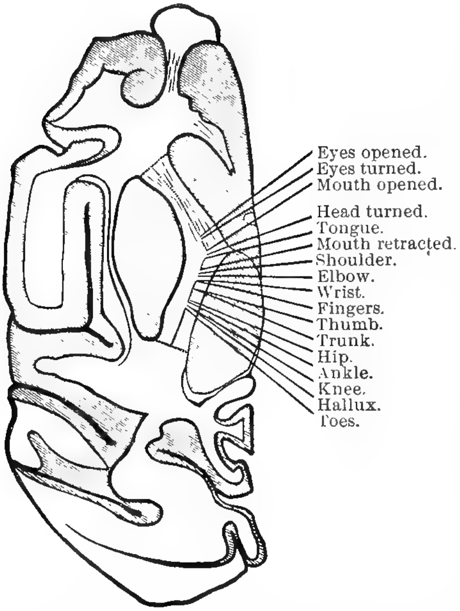


Horizontal section through the internal capsule: *Nc*, caudatum; *Nlf*, 1′, 2′, 3′, the three segments of the lenticula; *Tho*, thalamus; 2, tract of motor cranial nerves; 3, frontal pontile tract; 4, sensory tract; 5, anterior peduncle of the thalamus; 6, pyramidal tract. (Obersteiner.)

Functions of the External Capsule, Extreme Capsule, Claustrum, and Amygdala.—The functions of these parts are still prac-

tically unknown. The external capsule and extreme capsule doubtless contain fibres which connect the cortex with other parts, but their character and exact connections have not yet been determined. It is highly probable that the extreme capsule which lies close to the insula contains some of the tracts which run between the sensory and motor centres concerned with speech. It has been held that in the external capsule pass speech tracts from Broca's convolution to the bulb. The situation of the amygdala in the temporal cortex near the uncinate convolution indicates its probable relation to the function of smell; it has, in fact, been designated by Luys the olfactory ganglion.

FIG. 236.



Scheme of the arrangement of the motor fibres in the internal capsule. (Beevor and Horsley.)

Lesions of the Centrum Ovale.—Various terms, as centrum ovale and corona radiata, have been applied to the white substance of the hemispheres. The exact location of particular fasciculi of the projection, commissural, and association systems which are crowded into this region are gradually becoming more and more clearly defined, and to a certain extent these have been already considered, when discussing the sensory path, the pyramidal tract, and the various commissural and association systems, in previous sections in Chapter I. Lesions of special subdivisions of either the motor or the sensory projection systems will give symptoms similar to those produced by lesions of the areas or subareas of the cortex to which they come or from which they go. With reference to lesions of the projection fibres, one general diagnostic point of considerable importance is that the nearer the lesion is to the cortex the more likely it is to give rise to isolated or single symptoms, as to paralysis of a limb or portion of a limb, of the face or portion of the face, when the lesion cuts motor fasciculi; or to anesthesia limited to a segment of the limbs, trunk, or face, when the lesion severs sensory fibres; while, on the other hand, when the lesion is deeply situated near the internal capsule the complex of symptoms is more likely to constitute a hemiplegia or a hemianesthesia.

Lesions of the Motor Projection System in the Centrum

Ovale.—As shown when discussing the internal capsule, the bundles of the motor projection system are arranged in a regular order which has reference to the areas of representation in the motor cortex. It follows that those portions of the motor paths which lie between the cortex and the capsule must be arranged in a similar systematic manner. Subcortical motor symptoms will be produced by lesions situated in the white substance directly subjacent to the motor region,—in the irregularly wedge-shaped mass of fibres which pass from the posterofrontal and central convolutions in converging lines to the internal capsule. Irritative and destructive lesions in this region give a symptomatology which has some points of difference from that of the motor cortex. In favor of a strictly cortical or epicortical lesion of the motor zone are localized clonic spasm, epileptic attacks beginning with local spasm followed by paralysis, early appearance of local cranial pain and tenderness, and increased cranial temperature. In favor of a subcortical location of a tumor are local paresis or hemiparesis followed by spasm, predominance of tonic spasm, normal cranial temperature, and absence, slight degree, or very late appearance of local headache and of tenderness to percussion. (Seguin).

Lesions of the Sensory Projection Systems in the Centrum Ovale.—With regard to fillet radiations in the centrum ovale, it is possible for the purpose of gross study to locate at least three great sets of fibres: (1) the bundle which conveys the impulses concerned with so-called common sensibility, the fibres of this bundle, as already stated, probably approaching or reaching the cortex in the limbic lobe and posteroparietal region; (2) a bundle or set of fibres which proceeding from the lateral fillet turns downward so as to take its course to the superior temporal convolutions, conveying to them auditory excitations; (3) gustatory fibres which pass to the lower and more inferior portions of the temporal lobe, probably chiefly to the fourth temporal convolution. Lesions of the centrum ovale may therefore give rise to symptoms referable to either of these sets of fibres: to anesthesias of various sorts when the lesions are situated beneath the limbic and posteroparietal cortex; to word deafness or other interference with hearing when in the superior temporal subcortex; and to disorders of taste when in the inferior temporal subcortex. From the primary basal centres for vision have been traced bundles of fibres termed collectively the optic radiations of Gratiolet, which pass to the cuneus and its immediate vicinity, constituting the fibres of the visual projection system. Lesions of these fibres, like lesions of the cortical centres for vision, will produce hemianopsia or central visual defects or both. According to Henschen, analysis of all cases shows that the occipital visual path is in the ventral portion of the optic radiations, where it forms a bundle less than a centimetre thick. The path in a part of its course lies about

the level of the second temporal gyre and second temporal fissure. The macular fibres have a median situation, while the fibres of the dorsal retinal quadrant lie dorsally and those of the ventral retinal quadrant ventrally. A very limited lesion might therefore strike either of these sets of fibres, thus causing quadrant or sector hemianopsia, or disturbances of central vision. When with lateral homonymous hemianopsia the patient also has hemianesthesia, the lesion is probably in the tracts between the cuneus and the basal optic centres, being large enough also to involve the sensory tracts. Such a lesion would be best reached beneath the position where on the lateral aspect of the hemisphere the parietal, occipital, and temporal lobes come together. Hemianopsia is more likely to be complete when subcortical than when cortical. Lesions of the subcortex of the genual portion of the callosal gyre, gyrus fornicatus, and the uncinate and hippocampal convolutions, which contain most of the olfactory projection fibres, give rise to disorders of smell.

Lesions of Intracerebral Associating Tracts.—It is possible that we may in the future be able to give a symptomatology for lesions disrupting tracts connecting two convolutions, or one convolution with several convolutions of either the same or the opposite hemicerebrum. Lesions of Meynert's arcuate fibres, or *fibræ propriæ*, must cause special symptoms. Attention might be called here to a few of the known tracts or fasciculi for which a symptomatology should be sought. Vialet has shown that from the inferior calcarine (or lingual) region a distinct fasciculus, passing transversely outward beneath the occipital cornu of the ventricle, is distributed to the second and third occipital convolutions of the external surface. Beevor has determined in a marmoset another fasciculus from the calcarine region which passes forward into the centrum ovale and may connect this part of the cortex with other parts of the visual area, as with the supramarginal and angular gyres. Experiments of Schäfer and Barrett have shown that a connection exists between the excitable areas of the external occipital convolutions and a certain area for conjugate head and eye movements in the upper and middle frontal convolutions. Special forms of visual disturbance and some of the forms of transcortical aphasia are probably caused by lesions of these bundles. One great tract of arching association fibres first carefully studied by Meynert is the cingulum, which, with other well known association tracts, is shown in Fig. 73, page 62. Doubtless lesions of each of these sets of fibres give special symptoms. A lack of consideration of the commissural and association fibres is the source of much confusion in analyzing cases. Most lesions generally regarded as cortical involve to a greater or less extent the subcortex; and, as every convolution is connected with some other, and often with many others, association fibres must always be destroyed in these cases.

Microscopical Localization in the Cortex and Subcortex.—The discussion of functions and lesions of the cortex and subcortex has been chiefly with reference to gross changes and coarse functioning. The facts and views presented should prove of great value in localizing lesions of recognizable size and capable of causing symptoms of a more or less intense and special character; but many of the diseases and disturbances of the brain, whether classed under the general head of "mental" or "nervous," are wanting in specialized manifestations which can be easily referred to focal lesions. They give a symptomatology which is definite but diffuse, and which is not readily assigned to its anatomical and physiological bases. These mental and nervous affections include the cerebral neurasthenias, chronic affections, such as those due to alcohol and other toxic agents, and progressive degenerative diseases of various types. The cortex and subcortex must be searched by the diagnostician, and later perhaps by the histopathologist, for a rational explanation of the morbid phenomena; and this is a portion of the field of cerebral localization now being successfully entered with the aid of the new methods of research which have given us the wonderful revelations as to structure described in the section on the minute anatomy of the cortex. The changes in structure in these chronic, functional, and degenerative diseases remain for a long time, and in some cases always, of a microscopical character; and the microscope is always needed to interpret fully their true extent and significance. Horsley and Gotch, in studying the mode of action of a simple nerve centre in the spinal cord, showed that a distinctly measurable block to the transmission of a nervous impulse occurred between the time of the reception of an afferent impulse by a spinal centre and its discharge from the cells on the efferent side (see page 121), this block representing the time taken to traverse what is called the field of conjunction; that is, the region intermediate between the afferent or sensory and the efferent or motor spinal centres. Arranged in the cortex in stages or levels, as shown by Andriezen, is a series of nervoprotoplasmic plexuses which probably constitute cortical fields of conjunction. Exner's plexus represents one of these fields, and is probably the place of conjunction of the cerebral sensory and motor systems. In this as in other fields of conjunction the nerve fibres and the apical processes with which they come in contact become naked or noninsulated. Other fields of conjunction are represented by special structures in the polymorphic layer. In these plexuses, which Andriezen describes as extending sheet-like throughout the cortex, projection, association, and commissural fibres come in contact with the protoplasmic processes of cortical nerve cells. With the new methods of research it is now possible to see the gradual change and decay which impair or destroy cells as anatomical and physiological units, and also

disrupt their various connections and associations. Apical and basal processes and the collaterals and terminals which come in contact with them can be seen to pass through various stages of degeneration; cell bodies shrink and disappear in whole or in part; fields of conjunction are destroyed; and symptoms general and special arise. These minute changes of structure are the anatomico-pathological bases of such symptoms as amnesia, diminished power of attention and volition, diminished initiative and energy, diminished muscular power, blunting of the higher moral and ethical sense, insomnia, and serious disturbance in the balance between the cortical representation of the external world and the empirical Ego. (Andriezen.) The earliest changes in many if not in most of the chronic cases above mentioned are in the cortical fields of conjunction, the regions in which anatomical units or groups of them are brought together. These fields of conjunction become less and less permeable to all forms of excitation, and one of the first results is delay in reaction time. The association system, which gives way first in these cases, is probably represented most in the polymorphic layer of the cortex; it is the last and least organized, although subserving the highest functions, especially volition, and in chronic processes of disintegration it is the first to go.

Lesions of the Callosum.—Developmental arrests and lesions of the callosum occur with moderate frequency. Nearly a score of cases of tumor and rare instances of hemorrhage, softening, and other gross lesions of the callosum have been put on record. In idiocy the callosum has been found absent in whole or in part. Hochhaus collected records of twenty-four cases in which the callosum was absent. The symptoms of the recorded cases of lesion and arrest of the callosum have not been constant or distinctive, and the lesions have not been usually strictly limited to the callosum. Mott and Schäfer experimented with weak induction currents on the callosums of monkeys, and produced localized bilateral movements in all parts of the body. From before backward, in order, in different segments of the callosum, electrical stimulation caused movements of the head and eyes, the face, the shoulder, the trunk muscles, and the legs and tail. Cutting away one hemicerebrum, they found that by stimulating a thin middle strip of the callosum localized movements were produced on the side of the section, that is, on the side of the body opposite to that hemicerebrum with which the callosum was still connected, the order of movements being the same as above mentioned. Meynert, Obersteiner, and many others taught that the fibres of the callosum connect only identical areas of opposite hemispheres, but this is now disproved. The fibres of the callosum are extremely fine; and the new methods of staining have shown that many of them are only collaterals from axis cylinder processes of projection and arcuate fibre systems. Marchi's method in particular

has revealed widespread connections of the callosal fibres far different from those supposed to exist by Meynert. One or more convolutions in one half of the brain may be united through the callosum with one or many portions of the opposite hemicerebrum, according as these regions are connected in function. The old descriptions of the courses of many of the callosal bundles may, however, in a general sense be still regarded as correct: they diverge upward and downward and forward and backward into all or nearly all portions of the cerebrum, in the anterior part forming the anterior forceps, and in the posterior part the posterior forceps. According to Obersteiner, the callosum is the commissure *par excellence* of the cortex, cortex and callosum being usually wanting in equal degree; but Hamilton has advocated a different view, namely, that it is really a decussation of fibres arising from nerve cells in each hemisphere. These fibres after decussating run not into the cells of the opposite hemispheres, but directly downward into the internal capsule and parts below. Hamilton thinks it doubtful whether callosal fibres function in a direction downward, it being more probable that impulses pass along them from lower centres to higher. Sherrington and Muratoff both found that sometimes after lesion of the motor areas of the cortex—which Sherrington calls the "cord area"—they could trace secondary degeneration across the callosum into the opposite hemisphere. Having in regard the differing and newer views as to the course and functions of callosal fibres, it can only be said that lesions limited to the callosum would be likely to produce differing symptoms, not always of a positive character. Destructive lesions might give some bilateral paresis or incoordination affecting certain segments of the body according to the segment of the callosum invaded, in accordance with the determinations of Mott and Schäfer. Irritative lesions, as tumors, should give bilateral spastic phenomena or convulsions, and such convulsions have been noted in several reported cases. Hallucinations have also been noted. In a series of cases studied by Francis, the symptoms which seemed referable to the callosum were bilateral convulsions, stammering, uncertainty of gait, slowness of speech, change in temperament, strabismus, dilatation of the pupils, and rotation of the head. Bristowe, who is referred to by Bramwell as having written the most complete account of callosal tumors, gives the following as the progress of a case: "*first*, the occurrence of headache and other somewhat vague symptoms of progressive cerebral disease; *second*, the gradual onset of more or less well marked hemiplegia; *third*, the appearance in a greater or less degree of similar symptoms on the opposite side of the body; *fourth*, the coming on of dementia, with drowsiness, loss of speech, difficulty in swallowing, and want of control over the rectum and bladder."

Lesions of the Cerebral Commissures and of the Fornix.—The precommissure has been generally regarded as a structure connecting the opposite olfactory lobes and cephalic portions of the temporal lobes; but that it unites only homologous portions of the two hemispheres may now be regarded as doubtful. It is more probable that, like the callosum, its connections are of a more irregular and heterogeneous character. According to Hamilton, it may be doubted, with reference to the so-called interhemispheric commissures, whether these are "commissures" in the old sense of the word. The combined actions of the two halves of the body where they exist, as in the case of some of the cranial nerves, are attained by decussation of tracts of fibres, and not by union of corresponding nerve cells on opposite sides of the nervous axis. The facts recently demonstrated regarding the diverse connections of the fibres of the callosum have a bearing upon this view, and similar facts will probably be demonstrated sooner or later with reference to the other so-called cerebral commissures. Whatever may be the method of transmission of fibres through the precommissure, it is probable that some of them at least pass to the cortical organs of smell, so that lesions of this structure may cause olfactory disorders. The postcommissure is almost certainly in large part a decussation, some of its fibres going to the nuclei of the third nerve, and others passing to the quadrigeminal body. Presumably, therefore, lesions of this structure give rise to affections of equilibrium and of ocular movements, but clinical data regarding this matter are wanting. In part the postcommissure appears to be a decussation of the fillet, and its lesions therefore may also give rise to some sensory phenomena. Nothing that is known about the medicommissure can be applied in diagnosis. As already said when considering olfactory localization, Hill's view that the fornix is a part of the olfactory tract has much in its favor, and lesions of this structure should therefore give rise to olfactory symptoms.

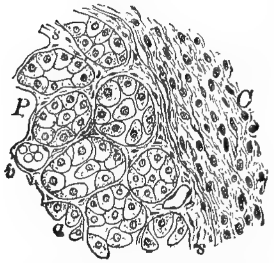
Lesions of the Ventricles.—The ventricles, their annexes, and their channels of intercommunication may be more or less occupied by lesions. Primary ventricular hemorrhage, which sometimes occurs but is rare, will be briefly discussed under encephalic hemorrhages. In other forms of cerebral hemorrhage, and especially in the common type from rupture of the lenticulostriate artery, the blood may break its way through the intervening tissues and inundate a part or the whole of the ventricular system, passing into the diacœle or third ventricle and by way of the iter into the fourth ventricle, causing much destruction in its course. Such a hemorrhage constitutes one of the most serious forms of cerebral apoplexy. Tumors may invade the ventricles. The symptoms in such cases will be largely those of the parts with which the growths are connected, or upon which they exert pressure, or which they irritate. The extent

to which these cavities may be occupied by neoplasms before death results is extraordinary. I have seen tumors, usually gliomata, nearly filling the metepicœle, the patients having lived for weeks or months. In one case in which a growth extended from the subgeminal region to one lateral lobe of the cerebellum, involving the dorsal and lateral aspects of the pons and oblongatas, the prepeduncle and the medipeduncle, the tumor was large enough nearly to fill the fourth ventricle. The patient lived for several months under my own observation, and had been attacked by intracranial disease a long time previous to my seeing him. Tumors and hemorrhages into the paracœles and diacœle may cause symptoms referable to the intraventricular striate body, the commissures, the fornix, the thalami, gemina, or callosum, according to their exact situation, extent, and character. Lesions may produce hydrocephalus by blocking the iter or the foramens of Magendie and Mierzejewski. Abscesses of the ventricles are occasionally observed. Hamilton holds that it is possible that the walls of the entire ventricular system are endowed with the power of indicating position. Experiments of Christiani and Bechterew seem to indicate that the gray matter of the third ventricle plays the part of a balancing organ. When the gray matter of this ventricle was injured, they found that the same difficulty from loss of the balancing faculty was experienced as when the semicircular canals are the seats of lesions. Hamilton, in the case of a patient who had the symptoms of Ménière's disease, at the autopsy found two tumors loosely attached to the choroid plexus, their free ends floating in the distended lateral ventricles. He thought it possible that the tumors dangling in the ventricular fluid may have stimulated the epithelial lining of the cavities and excited the vertiginous symptoms, as distinct evidences of disease of the ear were not present.

Lesions of the Hypophysis, or Pituitary Gland.—Numerous observations and investigations would seem to show that the posterior portion of the pituitary gland—that which chiefly contains the nerve tissues—is vestigial and probably without important function. On the other hand, the tendency of recent researches is to give the anterior portion some real functional importance. Vassale and Sacchi as the result of their experimental investigations in regard to the effects of destruction of the pituitary body conclude that although the symptom-complex following complete destruction of the pituitary gland offers analogies to that which follows extirpation of the thyreoid gland, they are unable to admit that the functional relations between these two bodies are such that one may, as Rogowitsch asserts, be a substitute for the other in the needs of the animal economy, and that as regards its functions the hypophysis belongs to the class of glands the destruction of which gives rise to the formation and accumulation

within the organism of special toxic substances. Considerable clinicopathological evidence would seem to place the pituitary body in relation with the disease known as acromegaly, and probably with other forms of dystrophy, such as the adiposis dolorosa described by Dercum. In nine out of eleven cases of acromegaly with autopsies, collected by Dana in 1893, the pituitary gland was enlarged. That it is sometimes diseased without the occurrence of acromegaly or other dystrophy, as in cases reported by Blackburn, does not preclude the idea that lesion of this organ may cause such affections; it may be that only certain perversions of the hypophysis give rise to such dystrophies. It has been thought that other glands may assume the functions of the pituitary body, but Oliver and Schäfer found that intravenous injections of pituitary extract markedly raised blood pressure, while thyreoid extract lowered it, and spleen extract at first lowered and then raised it. Slight enlargement of the gland has been known to take place in myxedema and in cretinism, and after extirpation of the thyreoid in animals. Gross lesions of the hypophysis are important independently of its functions, as they may give localizing phenomena because of its relations to neighboring parts, as the chiasm, the circle of Willis, olfactory bulbs and tracts, tuber, albicantia, and some of the basal sinuses and nerve tracts. Middleton Michel many years ago recorded a case in which the autopsy showed at the site of the hypophysis a tumor which had extensively invaded the adjacent soft parts. The patient complained of discomfort in the head and of cloudiness of vision, and was soon the victim of intermittent headaches. The imperfection of sight progressed rapidly until he was almost blind. The eyeballs preserved their parallelism, became sensitive to touch, and edema of the subconjunctival tissues was soon marked. He developed fever, insomnia, and delirium.

FIG. 237.

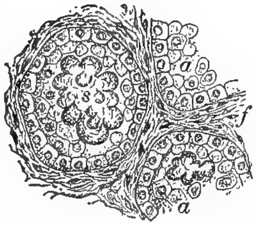


Section of human pituitary body: *C*, portion of posterior or nervous lobe; *P*, portion of anterior or glandular lobe; *a*, tubular acini; *s*, connective tissue septa; *v*, bloodvessels. (Piersol.)

Lesions of the Conarium or Pineal Gland.—Nothing need be said about the lesions of the conarium or pineal gland based upon its functions, as it is a vestigial structure and contains very little nerve tissue. It is, however, like the hypophysis, sometimes the seat of tumors, or it may be invaded by tumors or other lesions of neighboring parts. Coats has described an adenoid sarcoma containing small pieces of cartilage, and others have recorded the occurrence of other solid or cystic growths. Psammomata and small cystic growths are somewhat frequent. The smaller lesions, so far as known, give no recognizable symptoms. Large neoplasms originating in the conarium may give thalamic and quadrigeminal

symptoms, such as anesthesia, incoordination, interferences with sight and hearing, as well as symptoms which indicate hydrocephalus from blocking of the iter. Max Flesch has advanced the idea that the conarium in mammals is connected with the temperature apparatus, but this is not probable, although its lesions might cause temperature phenomena by invading the thalami or other thermic centres. The fibres of the pineal gland to the ganglion habenulæ, which are known to join the optic tract near the pregeniculum, Darkschewitsch regards as pupillary in function.

FIG. 238.



Section of human pineal body: *a*, *a*, acini; *s*, epithelium and calcareous concretions partially lining the acini; *f*, intertubular fibrous tissue. (Piersol.)

Lesions of the Tuber.—Polypnea is rapid or frequent respiration. Richet applied the term to a form of respiration which he observed in dogs exposed to a temperature of 86° F. The number of respirations suddenly increased from 350 to 400 per minute. The production of polypnea he believed to be a function of the oblongata. Its rôle is to regulate the temperature of the body, which is illustrated in the fact that an animal pants to cool itself. Richet called this function thermopolypnea, and believed it to be ordinarily a reflex function, but central when insufficient. Through it temperature is regulated by an exhalation from the skin or from the lungs. Ott found that when he cut the chiasm, punctured the tuber, or touched it just behind the chiasm, he always arrested polypnea and thermotaxis. Lesions of the tuber and its vicinity should therefore cause polypneic and temperature phenomena; but clinical data bearing upon this point are as yet wanting.

Lesions of Special Foci of the Basal Cinerea.—The optic tract receives a root from the region just behind the infundibulum. Hensen and Völckers have located the centre for movements of accommodation in the central gray matter of the floor of the third ventricle near the median line and just over the position of the albicantia; movements of accommodation were elicited by electrical stimulation at this point according to them, and of the iris by stimulation of a point a little farther caudad. Loss of accommodation they held was due to destruction of the centre for accommodation in this region, and loss of pupillary response to light, to lesion of the second more caudal nucleus. Spitzka and Westphal reported cases which in the absence of accommodative phenomena seem to confirm the conclusions as to the cephalic position of the centre for accommodation. In one case total and in the other considerable external ophthalmoplegia was present, accommodation being preserved in both. The nidi for accommodation were healthy. Berry and Bramwell have reported a case similar to those of Spitzka and Westphal, but without

autopsy. Borthen has reported a case of unilateral immobility of the pupil in which the pupil failed to respond to light, but accommodation was normal. The left pupil was dilated. On allowing light to fall into the left eye there was no responsive contraction; contraction by convergence and accommodation was present, but very slow. When the normal eye was covered the left pupil contracted down to a diameter of three millimetres. Reaction of the right eye was present, but reduced in the left. Borthen concluded that the affection might be due to a lesion of the nucleus for the constrictor of the pupil or of the fibres connecting the opposite pupillary nuclei or to spinal irritation. By exclusion he inferred that the symptoms were due to nuclear disease. I have had under observation a similar case of unilateral immobility of the pupil in a patient suffering apparently from disseminated gliosis or disseminated syphilitic disease of the cerebrospinal axis. These centres will be again considered when discussing affections of ocular movements due to nuclear and root fibre disease.

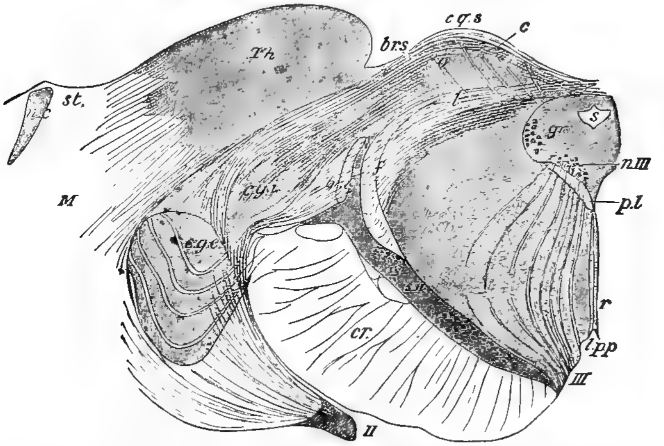
Functions and Lesions of the Quadrigeminal Body.—The exact functions of both subdivisions of the quadrigeminal body—the pregeminum and the postgeminum—have not been established to the satisfaction of physiologists and clinicians; but a practical unanimity of opinion has been reached as to the pregeminum being in some way associated with visual processes. Some authorities are inclined to regard the quadrigeminal body in man as an effete organ, but with Ferrier I believe that it has important functions in the human being, although these may be relatively much less important than in many lower animals. Ferrier holds that the quadrigeminal body is an essential portion of the mechanism which coordinates retinal and general sensory impressions with the mesencephalic motor apparatus of equilibration and other adaptive reactions. In proportion as the higher cerebral organ for vision increases in the process of development, the quadrigeminal body seems to decrease, although it certainly continues to retain active functions connected with vision and station in man. Disorganization of the ganglia in rabbits caused blindness, dilatation, and immobility of the pupils, and marked disturbance of equilibrium and locomotion. The pregeminum is certainly connected with the occipital cortex by special bands of fibres,—the optic radiations. The optic lobes of fishes, which correspond with the pregeminum, are directly connected with the optic nerves and are the chief central organs of vision in these animals; but, on the other hand, it must be remembered that the quadrigeminum is highly developed in lower animals in which the eyes and vision are in a very rudimentary state; so that it is probable that only certain portions or layers of the pregeminum have visual functions. Entire removal of the optic lobes of fishes by Ferrier rendered station and locomotion

impossible. Mechanical and electrical stimulation of the quadrigeminum caused more or less coordinated movements which sometimes took the form of spastic phenomena. Electrical stimulation of the pregeminum on one side in monkeys produced almost always dilatation of the opposite pupil, this being soon followed by dilatation of the pupil on the same side, also by elevation of the eyeballs upward and to the opposite side, with numerous other motor manifestations. Bechterew with others believes that the postgeminum has distinct auditory functions and is also a motor or coordinating centre. He has traced the lateral or inferior fillet, which lies to the outside of the general fillet, from the apex of the superior olive to the nucleus of the postgeminum, and this lateral fillet is without doubt the most important basal tract for hearing. Irritation of the postgeminum of the dog by Ferrier caused a short bark or cry and every variety of vocalization; and Goltz and Steiner believe that the postgeminal tubercles are centres for emotional manifestations, because in frogs the croaking cries in response to sensory irritation cease after their extirpation. In a case reported by Berry and Bramwell, in which the lesion was probably subgeminal, screaming fits occurred without apparent cause, and sometimes lasted half an hour, and these screaming fits were compared by them to the cries which electrical irritation of the postgeminum produces in lower animals. Lesions of the quadrigeminum usually implicate both the pregeminum and the postgeminum, and therefore may give disorders of vision and hearing associated with those of equilibration. If restricted to the pregeminum, affections of vision and of ocular movements are most prominent. Trismus with opisthotonos, disordered or reeling gait, nystagmus, and special affections of the oculomotor, pathetic, and abducens nerves have been described in recorded cases.

Subthalamic and Subgeminal Lesions.—A macroscopic lesion of the subthalamic region gives a complicated symptomatology, but one which will eventually be recognized in its details and properly referred. Edinger truly says of this region that its lesions involve such a tangle of different sorts of fibres that their symptoms are of the most manifold description, and a positive diagnosis can hardly be made. Foci of disease in the vicinity of the crus implicate motor fibres to the opposite side of the body. Sensory and vasomotor affections and oculomotor disturbances may also be present. With simultaneous paralysis of one oculomotor nerve and of the opposite side of the body a lesion under the quadrigeminum is probable. "Such patients have either wholly or in part lost control of the limbs on one side of the body, the upper lid droops, the pupil is dilated, and the whole eye turned outward by the rectus externus. The same symptoms might come from a tumor at the base of the brain. It is therefore important for diagnostic purposes to know whether the ocular paralysis appeared simultaneously with

the paralysis of the extremities,—a condition of things which could only very rarely occur in the case of a tumor at the basis cerebri. If anesthesia appears it is only present on the side opposite the disease. The sensory fibres run to a great extent in the lemniscus." (Edinger.) The red nucleus might be affected, and its connections which have been in large part traced are such as to indicate that it has some relations to equilibration. Some of the most important of these connections are with the thalamus, the lenticula, and the prepeduncles. Bechterew found the substantia nigra markedly degenerated and shrunken as a result of destruction of parts of the basal ganglia, and he believes that this structure is connected with the striata. Witkowski has also described descending degeneration

FIG. 239.



Section across the pregeminum and the adjacent part of the thalamus: *s*, Sylvian aqueduct; *gr*, gray matter of aqueduct; *c.q.s.*, quadrigeminal body, consisting of, *l*, stratum lemnisci, *o*, stratum opticum, *c*, stratum cinereum; *Th*, pulvinar of the thalamus; *c.g.i.*, *c.g.e.*, internal and external geniculi; *br.s.*, *br.i.*, superior and inferior brachia; *f*, upper fillet; *p.l.*, dorsal longitudinal bundle; *r*, raphe; *III*, third nerve; *n.III*, its nucleus; *l.p.p.*, postcribrum (posterior perforated space); *s.n.*, substantia nigra, above this tegmentum with its circular nucleus; *cr*, crus; *II*, optic tract; *M*, medulla of hemisphere; *n.c.*, caudatum; *ts.*, stria terminalis. (Quain-Meynert.)

implicating the substantia nigra. The complicated structures of the subthalamic and subgeminal regions are in part shown in Fig. 239, and comparison should be made with Figs. 76 and 77 on page 66.

Lesions of the Crus.—The chief result of a destructive gross lesion of the crus is the production of alternate hemiplegia of the oculomotor type. From involvement of the descending pyramidal tract result paralysis of the leg, arm, and lower face of the opposite side, and, through implication of the third nerve at or near its superficial origin, paralysis of the part supplied by this nerve on the same side as the lesion, giving ptosis, dilated pupil, and paralysis of all the ocular muscles except the external rectus and superior oblique. Occasionally lesions in this position cross the median line

and give a complex of symptoms due to irregular bilateral involvement of the pyramidal tracts and the third nerves. Some cases of oculomotor paralysis combined with monoplegia or hemiplegia present special features which make focal diagnosis difficult. The oculomotor paralysis may be on the same side as that of the extremities and face; and in such cases, if the history is of successive attacks,—and occasionally even when it is not,—the two sets of symptoms may be due to different lesions. This is perhaps the best explanation of the majority of such cases, which are usually syphilitic. A lesion of irregular shape, chiefly on one side, but crossing the median line in the region of the root fibres of the third, might involve both the crus and the root fibres of one or both third nerves in the subthalamic or subgeminal region, other intermediate parts, such as the red nucleus, subthalamus, deep transverse fibres, and substantia nigra, not giving known symptoms. The fillet might escape, owing to its more lateral position.

FUNCTIONS AND LESIONS OF THE CEREBELLUM.

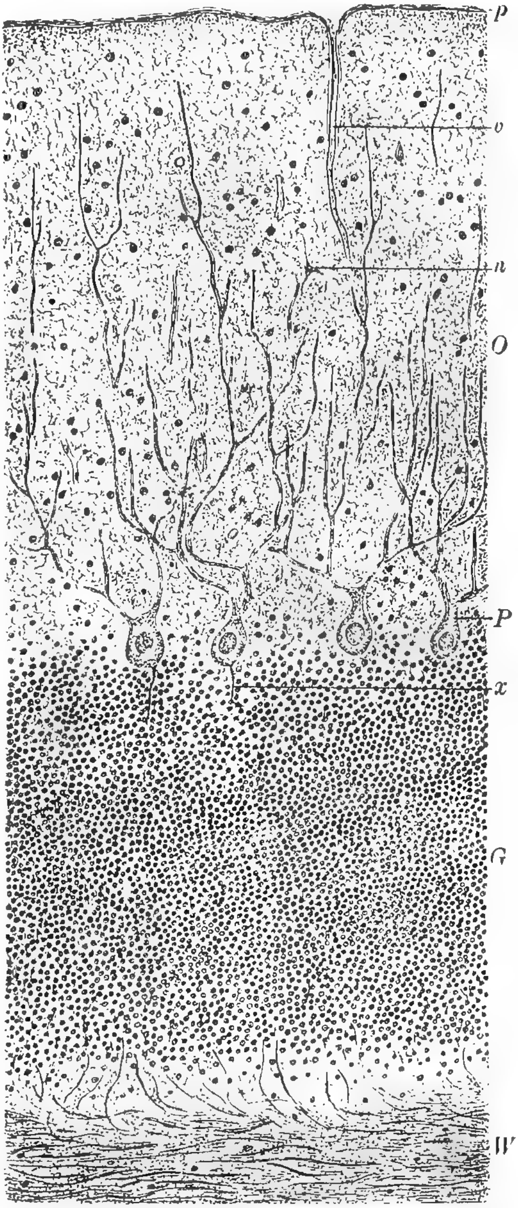
Anatomical Relations of the Cerebellum.—The gross anatomy and anatomical relations of the cerebellum have already been sufficiently described (see pages 67 and 73). The conclusions arrived at by Marchi, Bruce, and others, given there with reference to the cerebellar conducting tracts, are largely but not fully in accord with those of other investigators; but the discrepancies are not sufficient to affect clinical deductions. The connections of the cerebellum are practically as given in the descriptions on pages 107 to 109, and as shown in the diagrams of Bruce and Van Gehuchten, but it must be borne in mind that the relations and interrelations of the cerebellum are not yet thoroughly known. The results obtained by all experimentalists and histologists agree in the main, and show that the cerebellum has traceable connections with the prefrontal lobe, the occipital and temporal lobes, the red nucleus, and probably also with the thalamus; with the quadrigeminal body, the pontile nuclei, the inferior olives, certain nuclei and fibre tracts of the oblongata, and with special regions of the spinal cord. Nearly all observers have confirmed one another as to the connection of one lateral lobe of the cerebellum with the opposite olive, this tract constituting a great efferent system, the cerebello-olivary system. Meynert first described atrophy of the inferior olive in association with atrophy of the opposite cerebellar hemisphere. Von Gudden and Vejas found that the cerebello-olivary tract underwent atrophy after removal of a cerebellar hemisphere in new-born animals; and Bechterew and Bruce, Darkschewitsch and Freud have described myelination as occurring only in the last month of intra-uterine life, by which time the other constituent tracts of the restiform body are fully medullated. These observations therefore

confirm one another and prove beyond a doubt the intimate anatomical and functional relationship between the lateral lobe of the cerebellum and the opposite inferior olivary body. Ferrier and Turner's experiments, in connection with facts recorded by other observers, support the view that the internal subdivision of the restiform body is an efferent cerebellar tract which goes to Deiters's nucleus, this nucleus being an internode between the cerebellar and the spinal system. In two cases in which the lateral lobes of the cerebellum were removed by Ferrier and Turner degeneration was found in the spinal cord, especially in the anterolateral regions.

Minute Anatomy of the Cerebellar Cortex.—The cerebellum, whose functions have always been more obscure than those of the cerebrum, has also, like the latter, been subjected to the newer histological methods of investigation. The cerebellar cortex is usually described as composed of (1) an inner reddish gray or granular layer, (2) an outer gray or molecular layer, and between these (3) a row of large flask-shaped cells known as the cells of Purkinje. Fig. 240 shows this arrangement in the human cerebellum, while Fig. 241 is a skeleton drawing by Berkley of a cerebellar leaflet from the brain of a dog, the appearances having been built up from sections variously stained. The granular layer or zone is narrow at the base of the leaflet, gradually spreading out until its breadth is several times as great as at the base. Berkley describes no less than six different varieties of cellular bodies in the granular zone. Some of these forms are recognized by Piersol and others as most important, these being (1) small round multipolar cells with little protoplasm and large nuclei ; (2) larger multipolar cells, few in number, and resembling somewhat the cells of Purkinje. The axis cylinder processes of the small cells pass to the outer layer. The axis cylinder processes of the large cells and the protoplasmic processes of both form arborizations within the granular layer. The cells of Purkinje are among the largest ganglion cells of the body, recalling the great pyramidal cells of the cerebrum, but presenting striking differences. Each cell possesses a large nucleus and also a nucleolus and an axis cylinder process which passes down into the white substance to become a medullated nerve fibre. Their protoplasmic processes are of great extent and interest, their direction in general being towards the cerebellar surface, as shown in Figs. 240 and 241. The numerous ramifications of these processes are not in all directions, but, as Obersteiner says, are like those of an espalier fruit tree, in two directions only, so that in a section across a convolution the processes show a fanlike radiation, while in a section cut lengthwise they show simply as an edge. The Purkinje cells form a great connecting link between the molecular and the granular layer. They are enveloped in a feltlike capsule or basketwork of fibres derived

from surrounding cells. The encapsulation of the Purkinje cells gives a lymph space of great extent to the outer layer. Beyond the Purkinje cells, towards the surface, is a considerable number of small rounded and angular multipolar cells, the largest of which are undoubtedly nerve cells. In the molecular layer, in a fine reticulated supporting neuroglia, besides the cells with their encapsulating basketlike tufts above described, are the elaborate arborizations of the protoplasmic processes of the Purkinje cells, and also small multipolar cells whose branched processes extend peripherally, while other axis cylinder processes extend centrally but are probably confined to this layer. In the outer zone of the molecular layer a few horizontal medullated fibres are seen. The nuclei of the neuroglia are seen distributed in this layer throughout its central and mid regions, becoming less obvious towards the pia, and in some places being entirely absent. On the outermost border of the cerebellar leaflet subjacent to the pia is a condensation of substance known as

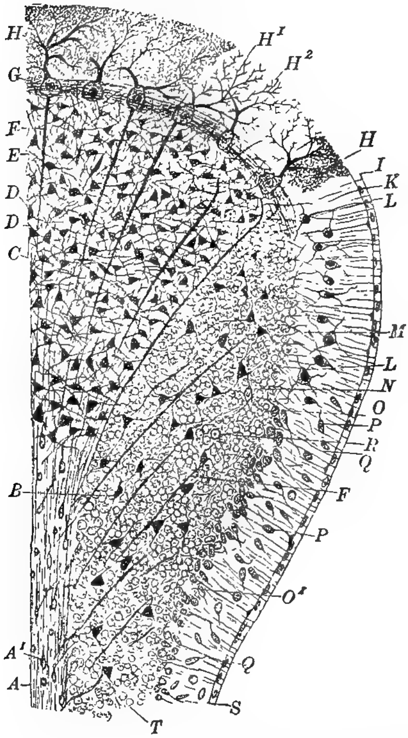
FIG. 240.



Section of human cerebellum: *W*, white matter; *G*, granule layer; *O*, molecular layer; *P*, cells of Purkinje; *x*, axis cylinder processes passing into granule layer; *n*, small nerve cell; *v*, pial bloodvessel; *p*, pia. (Piersol.)

the *limitans externa*, which is made up of a feltwork of fine glia fibrils derived from oval and pyramidal nuclei. Outside this limiting membrane is a subpial lymph space of considerable magnitude, crossed by connective tissue fibrils which come from the innermost layer of the pia and go into the cerebellar substance. Here and there in the central white matter multipolar cells are found; otherwise the white substance is like that of the cerebrum. From the white substance one set of fibres can be traced to the Purkinje cells; another set passes to Dennissenko's eosin cells in the granular layer; some of this set probably come in contact with other fibres before touching the eosin cells, some exceedingly fine but medullated fibres passing on to the molecular layer. Still another set of fibres, much smaller in number and non-medullated, passes to the inferior and middle thirds of the molecular layer. Berkley makes an effort to separate the cerebellar fibre systems into efferent and afferent according to their connections and histological peculiarities.

FIG. 241.



Skeleton drawing of half a cerebellar leaflet, built up from sections stained by various recent methods: *A*, core of medullated fibres; *A'*, glia nuclei among them; *B*, layer of granule or hematoxylin cells; *C*, granule layer with eosin cells, straight nerve fibres running to the Purkinje cells, and the anastomosing fibre plexus; *D*, *D*, eosin cells; *E*, fibre plexus; *F*, *F*, straight fibres running through the granule layer; *G*, tangential fibres at the outer edge of the granule layer; *H*, *H*, Purkinje bodies with outspreading branches; H^1, H^2, turning of the terminal filaments beneath the pia; *I*, pia; *K*, nuclei in the subpial space; *L*, *L*, small nerve cells in the inferior third of the molecular layer; *M*, fibres radiating out of the granule layer; *N*. nerve cells in the granule layer; *O*, row of pear-shaped and round nuclei at the outer edge of the granule layer; *O'*, Purkinje cell space, capsule not drawn; *P*, *P*, glia elements of the molecular layer; *Q*, *Q*, fibrillæ passing off from the pear-shaped cells attached to the limitans externa; *R*, supposed glia nucleus in the granule layer; *S*, limitans externa; *T*, granule nuclei. (Berkley.)

Physiological Theories and Facts regarding the Cerebellum.—The size and structure of the cerebellum in all vertebrate animals indicate that its functions are important. Animals which swiftly and rapidly coordinate their movements have cerebellums of large size and complex development. Edinger has shown that osseous fishes, which are excellent swimmers, have cerebellums enormously developed as com-

pared with those of the cartilaginous fishes, which live tranquilly in the deep. The lateral lobes of the cerebellum, so largely developed in man and the higher animals, are practically wanting in amphibia and reptiles. A view commonly held, which seems to have originated with Nothnagel, that the vermis or middle lobe is the only part of the cerebellum with determinable functions, can no longer be regarded as correct. Histological studies and the experiments of Luciani and of Ferrier and Turner show that the lateral lobes, as well as the vermis, have important functions, and that their lesions should give recognizable symptoms. Gowers holds that while the middle lobe probably presides over the coordination of movement, it may act on the opposite cerebrum and other parts through the cortex of the lateral lobes. According to Berkley, the nerve elements of the cerebellum observed as a unit bespeak a sensory and not a motor organ, the great cells of Purkinje having a closer likeness to those of Clarke's column than to any other bodies in the nervous system; but arguments as to function based upon the size and character of the cells must be received with caution. The cerebellum early attracted attention on the part of physiologists, as Flourens, Magendie, and Weir Mitchell, and has been the subject of continuous investigation. Some of its recent investigators are Luciani, Marchi, Borgherini and Gallerani, R. Russell, and Ferrier and Turner. The facts elicited by all are practically the same, but different inferences have sometimes been drawn, as by Luciani and by Ferrier, although this in some instances would seem to have been due to different methods of interpreting phenomena, as when one observer describes an animal as rolling away from and the other as rolling towards the side of the lesion made in the cerebellar peduncle. Borgherini and Gallerani found that lesions profoundly affecting the cerebellum produced symptoms resembling those of locomotor ataxia, and that a superficial lesion of the organ, which in experiments falls of necessity on its dorsal and caudal aspect, gave as a constant and permanent result a tremor of the head and neck. They also found that injury to the cerebellum caused trophic disturbances, but was not accompanied by any modification of muscular force nor by any alteration of special or general sensibility. One of the differences between Luciani and Ferrier is as to the *immediate* effects of cerebellar lesion. Luciani regards these as irritative, while Ferrier describes them as dynamic or inhibitory. These immediate effects have been described in much the same way from the days of Flourens and Magendie to the present time. In dogs, as detailed by Luciani, they are disquietude, frequent howls, pleurothotonos, tonic extensions of the anterior extremities, and a tendency to roll from one side to the other. After total extirpation of the cerebellum, the symptoms are bilateral, and pleurothotonos is replaced by opisthotonos, and in monkeys the tonic extensions of the extremities are

replaced by tonic flexion. Experimenters are in general accord in stating that lesions of the cerebellum do not produce sexual or psychical manifestations, nor discoverable impairment of special sensibility nor of any of the forms of cutaneous or muscular sensibility. An important fact is that one half of the cerebellum exerts its influence on the same side of the body as itself, its action on the spinal cord being direct and not crossed. According to Luciani, the permanent effects of cerebellar ablation—the effects which persist after the irritative phenomena have subsided—can be arranged in three classes, which he describes as (1) asthenic, (2) atonic, and (3) astatic, regarding the three as simply external phenomena of one internal morbid process. The animals showed extraordinary disturbance of station and locomotion, and long persistence of unsteadiness of the trunk and limbs upon effort. According to Ferrier, the animals operated upon retained muscular strength, Luciani believing, however, from his own experiments that this was much impaired. Luciani also differs from Ferrier with reference to tonic contractures, believing them to be present.

Lesions of the Middle and of the Lateral Lobes.—Complete removal or large lesion of the vermis produces practically the same symptoms as ablation of the entire cerebellum or of its lateral lobes, but, unlike lesion of one lateral lobe, it does not affect one side of the body more than the other, and its effects are more pronounced in the head and trunk than in the limbs. Extirpation of this lobe is followed by secondary degeneration of both postpeduncles, which has been traced to the nucleus of Deiters. Bruce and Bechterew have traced connections between this tract, called by Edinger the direct sensory cerebellar tract, and the roof nucleus of the opposite side. In a case of gliosarcoma recorded by de Michele, chiefly involving the postvermis or inferior vermis, the symptoms properly referable to the cerebellum seemed to be progressive weakness of the extremities, general impairment of nutrition, unconscious yelling or screaming, and lateral curvature of the vertebral column. The patient also showed other general pressure and invasion symptoms of brain tumor. While it is true that disease of one lateral lobe of the cerebellum has been recorded as having no symptoms, such records are probably indicative of lack of close observation, although a slowly growing cerebellar tumor may for a long time cause scarcely detectable phenomena. After destruction of the lateral lobe, animals exhibit great unilateral disturbance of station or locomotion, and unsteadiness of trunk and limbs on muscular effort, on the side on which the operation has been performed. According to Luciani, both muscular strength and tone are impaired; but Ferrier does not give these meanings to the phenomena, regarding them rather as phenomena of incoordination. He records that knee-jerk was either not impaired or was

increased on the side of the lesion. The astasia or weakness, whichever it is, is usually more marked in the upper extremities. In a case of fibrosarcoma of the left cerebellar hemisphere recorded by Sobotka, the symptoms referable to the cerebellum were inability to walk for any length of time, enormously exaggerated knee jerk, and marked foot clonus, with right facial paresis, all the manifestations being slightly more marked on the right than on the left side. In a case of echinococcus of the left lateral lobe reported by Sonnenburg, the symptoms were sleepiness, loss of coordination, diminished vision, and difficult speech, the patient also suffering from violent headache, vertigo, and disturbance of respiration.

Lesions of the Cerebellar Peduncles.—The symptoms following division of the peduncles are very similar to those occurring after removal of the lateral lobe, the chief differences being a tendency on the part of the animals to roll around their longitudinal axes,—according to Magendie, Ferrier, and others, towards the side of the lesion, according to Luciani, towards the opposite side. Bannister has reported a case of hemorrhage into the ventral surface of the prepeduncle, which caused sudden disturbance of equilibrium, vomiting, with possibly contralateral paresis, also lateral decubitus on the side of the lesion and drawing of the head to the right side, and rigidity of the left arm. After section of the prepeduncle and velum a tract of degeneration can be traced backward into the vermis. According to Ferrier and Turner, the chief fact brought out in their study of degeneration in the sphere of the prepeduncle is that the brachium conjunctivum is efferent in function and undergoes complete degeneration after extirpation of the lateral lobes. Cases of irritative lesion of the medipeduncle have been reported in which the body has shown a tendency to gyrate involuntarily around its longitudinal axis, that is, to produce this form of forced movements; but this symptom may be absent in destructive lesions. In the case of an irritative lesion the patient may have an irresistible impulse to lie on one side. Some cases of idiocy with whirling tendencies probably have irritative or atrophic lesions of this peduncle. The medipeduncle is more frequently the seat of separate disease than either of the others. The postpeduncle has an external and an internal division, the external being connected with the spinal cord through the direct cerebellar tract, also with the clavate and cuneate nuclei and with the opposite inferior olive; the internal division is connected with the so-called auditory nuclei. Ferrier and Turner found that degenerations following extirpation of the lateral lobes were limited to the external division, and that those following extirpation of the middle lobe were limited to the internal division. A certain attitude after section of the postpeduncle was constant and characteristic, viz., curvation of the vertebral axis, with the concavity towards the side of the lesion, adduction and flexion of the

limbs on the side of the lesion, and abduction and extension of those on the opposite side. The direction of deviation of the chin was usually to the side opposite to the lesion. In several cases, after division of the postpeduncle, anesthesia of the cornea on the side of the lesion was observed, due to implication of the immediately subjacent secondary descending (usually called ascending) trigeminal root.

The Flocculus.—Some investigations and observations have shed a little light upon the functions of the flocculus. Edinger, for example, has traced fibres from the flocculus into the nucleus of the abducens or external oculomotor nerve of the opposite side, but Bruce has not been able to trace this connection. As bearing upon this relation, the fact that in a very few instances nystagmus has been reported as associated with disease of the flocculus is of interest. The flocculus appears very early in the development of the cerebellum. Mihalkovics states that it appears contemporaneously with the vermis. The fibres which form its core or peduncle begin to medullate during the fifth month. According to Bruce,

FIG. 242.

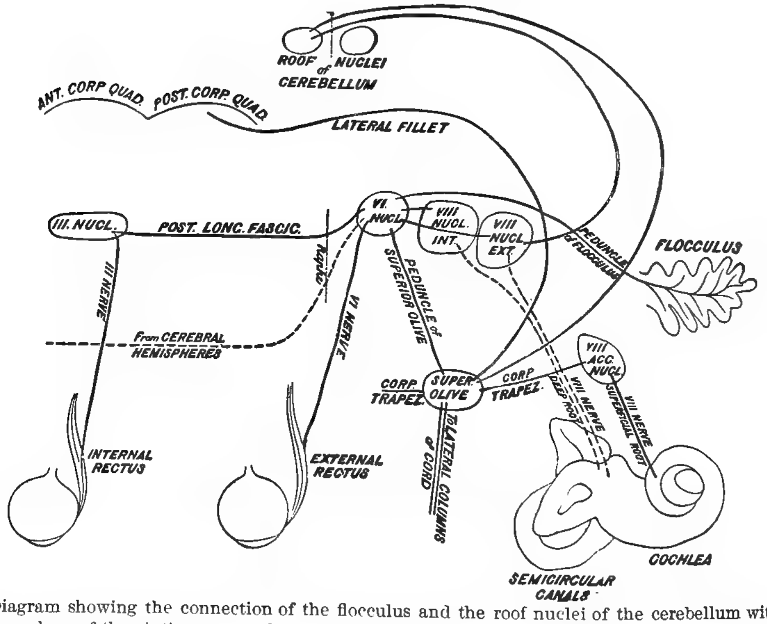


Diagram showing the connection of the flocculus and the roof nuclei of the cerebellum with the nucleus of the sixth nerve and other parts, and also other connections of the sixth nerve. (Bruce.)

successive transverse (frontal) sections show that this peduncle passes inward towards the lateral angle of the fourth ventricle, and that the majority of its fibres pass inward and enter into relation with both the external and internal nuclei of the auditory nerve

and with the nucleus of the sixth nerve on the same side. It is also connected with the accessory nucleus, and probably with the dentatum. Bruce believes that the associations of the flocculus point to it as an important structure related to the vestibular branches of the auditory nerve, which are probably not true nerves of hearing, but rather nerves of space. The connections of the flocculus are indicated in the diagram, Fig. 242.

The Clinical Phenomena of Cerebellar Disease.—A careful study of a large number of cases of cerebellar disease shows that the most important symptoms and those of most frequent occurrence are headache, vomiting, vertigo, double optic neuritis, unsteadiness of station or gait, tonic spasms or contractures, which may be either paroxysmal, continuous, or nearly continuous, tremor sometimes associated with voluntary movements and seen oftenest in the upper extremities, pleurothotonos, opisthotonos or retraction of the head, and paresis or paralysis of the extremities or of the cranial nerves, usually of one side. The symptoms in carefully recorded cases are, on the whole, in agreement with the results of physiological and histological research, although they may seem at times to conflict. This apparent conflict can be explained. The symptoms are not always to be directly referred to the cerebellum; they may be due to irritation of the bone or of the membranes, to pressure upon the adjacent parts, to acquired hydrocephalus, and to the reflection or diffusion of irritation, as well as to the cerebellar disease itself. The extreme irritation of the dura in most cerebellar tumors probably chiefly accounts for the headache, as even this symptom is occasionally absent when the neoplasm is small and develops within the substance of the cerebellum, as in some cases of glioma. Vomiting and vertigo result from dural irritation, but in cerebellar cases they seem sometimes to have special peculiarities and to be unusually severe. They may be due sometimes to the more or less direct irritation of the pons and oblongata. Disturbances of pulse, respiration, and temperature not dependent upon the site of the lesion may be present. A large cerebellar growth, hemorrhage, or abscess may give manifestations due to pressure upon the quadrigeminal body, pons, oblongata, or cranial nerves. Paresis or paralysis of the extremities has been noted as occurring both on the side of the lesion and on the opposite side. This may be accounted for by pressure on the pyramidal tracts of either side. When loss of power is noted as having occurred on the same side, it is probable that the symptom is of the nature of the ataxia or motor impairment which has been described as occurring in animals. The irregularity in the cranial nerve phenomena can be best accounted for as the effect either of pressure or of invasion. Hydrocephalus, which is acquired in many cases of cerebellar lesion, may give special features to the symptomatology. The apathy or stupor sometimes described, and

the affections of the optic and auditory nerves, may in some instances be attributable as much to the hydrocephalus as to the original disease. Knee jerk is commonly affected in cerebellar disease. Cases reported by Dercum and others show that it may be differently affected: it may be absent; it may be absent at one period of the affection and present at another; it may be exaggerated; it may be different on the two sides; or, as in one case of my own, it may be possible to elicit a crossed knee jerk. The difference between the effects of destructive and irritative lesions is important in endeavoring to fix the exact site of a cerebellar lesion: thus, destruction of the cephalic portion of the vermis causes a tendency to fall forward, and irritation a tendency to fall in the opposite direction. Destruction of the caudal portion of the vermis, on the other hand, causes a tendency to fall backward, while irritation will bring about the muscular adjustment necessary to counteract this tendency. In like manner a destructive lesion in the lateral lobe may cause a tendency to move in a direction opposite to that produced by an irritative lesion. It is probable that the effects observed in man are more frequently due to destruction of tissue than to irritation. Loss or impairment of sensation in the distribution of the sensory portion of the fifth nerve has sometimes been observed as the result of operation on the cerebellar peduncles. Ferrier and Turner during operations on the medipeduncle and postpeduncle found anesthesia and subsequently some degeneration of the trigeminal nucleus. These results were undoubtedly due to lesions of the long descending branch of the fifth nerve, usually known as the ascending branch.

Cerebellar Affections of Congenital Origin.—A few important cases of cerebellar atrophy or deficiency of congenital origin have been recorded. An old and remarkable case, reported by Combette, is that of Alexandrine Labrosse, a girl who lived to the age of eleven years. Her cerebellum was completely atrophied, its place being occupied by a cyst. She was five years old before she was able to stand, and at the age of seven was very insecure upon her legs. Shuttleworth and Taylor and Ferrier have recorded another remarkable case of an imbecile girl who died at the age of fifteen years. No deficiency existed in her sensory faculties, but she showed a general muscular weakness and tremor in her hands when she was using them, which was attributed to debility associated with a phthisical condition. She could walk well and steadily, though she was never known to run. The cerebrum was well developed, but the cerebellum was of the most diminutive character. Nonne and Marie and others have described a form of hereditary cerebellar ataxia associated with atrophy of the organ and of the spinal cord. The peculiar features of this form of disease will be discussed in connection with the affections with which it may be confounded.

CRANIOCEREBRAL TOPOGRAPHY.

Definitions.—Craniocerebral topography is the art of determining the relations of certain parts of the brain to definite points on the skull. Its chief practical purpose is to assist the surgeon in trephining for such affections as intracranial hemorrhage, tumor, abscess, concealed fracture, sinus thrombosis, mastoid abscess, disease of the Gasserian ganglion, purulent leptomeningitis, idiocy, epilepsy, and hydrocephalus. Anatomists and surgeons have employed various methods of determining craniocerebral relations. Cunningham hardened the brain in the skull, removed portions of the cranial walls, and made casts of the preparations. In Figures 243 and 244

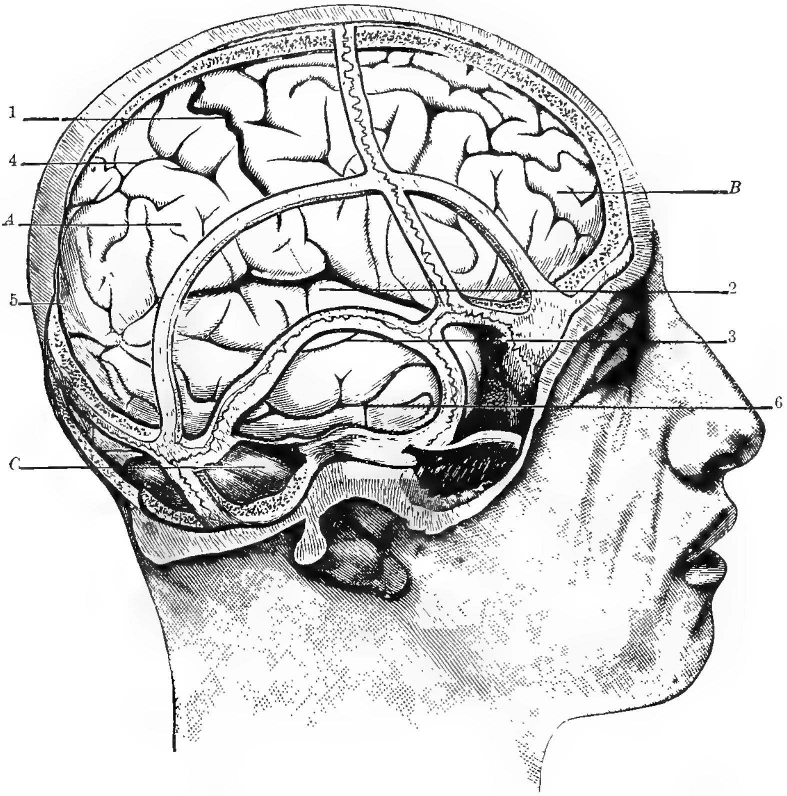
FIG. 243.

Drawing of a cast of the head of a newborn infant: 1, central fissure; 2, fissure of Sylvius; 3, supertemporal fissure; 4, parietal (intraparietal) fissure; 5, parieto-occipital fissure; *A*, position of parietal eminence; *B*, position of frontal eminence. (Cunningham, in Morris's Anatomy.)

are shown drawings of specimens prepared in this way, which indicate not only the position of the sutures with reference to the parts beneath, but also some of the most important lines and points on the skull. On page 147 (Fig. 134), numerous skull landmarks, mainly after Broca, have been given in connection with a consideration of craniometrical investigations. Some of these landmarks are used by the surgeon in determining proper points for trephining, as, for instance, the glabella, inion, bregma, vertex, auricular point or ex-

ternal auditory opening, nasion, and stephanion. Other points or lines are the external angular process of the frontal bone, the parietal eminence, the orbital arch, the zygoma, the mastoid process, and the superior arch of the occipital bone. The glabella, as already defined, is the triangular space between the eyebrows, and the inion is

FIG. 244.



Drawing of a cast of the head of an adult male: 1, central fissure; 2, fissure of Sylvius; 3, supertemporal fissure; 4, parietal (intraparietal) fissure; 5, parieto-occipital fissure; 6, meditemporal (second temporal) fissure; *A*, position of parietal eminence; *B*, position of frontal eminence; *C*, lateral sinus. (Cunningham, in Morris's Anatomy.)

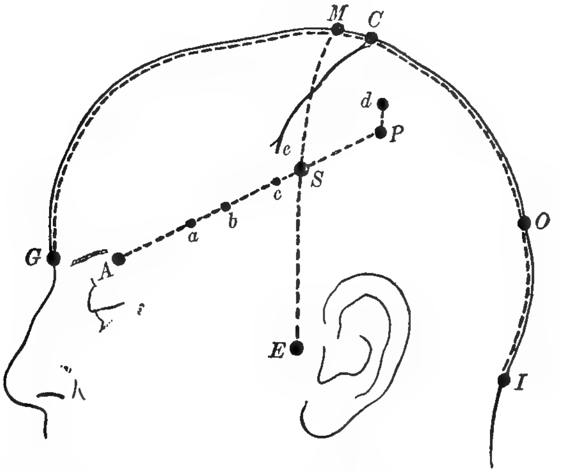
the external occipital protuberance. Usually the cephalocaudal or anteroposterior line of the skull is taken from the glabella to the inion. The stephanions, superior and inferior, are the intersections of the coronal suture with the ridges for the temporal fascia and the temporal muscle respectively. The bregma is at the junction of the coronal and sagittal sutures, and the vertex is the highest point of the skull, a little caudad of the bregma. The auricular point corresponds to the centre of the external auditory orifice, and the nasion, which is close to the glabella, to the junction of the nasal and frontal bones. The parietal eminence, the most prominent part of

the parietal bone, is often referred to, and is quite evident in the child, but frequently in the adult it is obscure and varies somewhat in position. It is necessary to take it into account, as it enters into the calculations of surgeons in defining certain lines or points for operation. The external angular process of the frontal bone is at its junction with the external portion of the orbit. The line of the orbital arch reaches from the external angular process of the frontal bone to the internal angular process. The zygoma is the narrow projecting bar of the inferior external border of the temporal bone. The mastoid process, which is the nipple-shaped process of the temporal bone behind the ear, is the point of departure for numerous measurements, and is also the position for certain operations. The superior curved line of the occipital bone, or superior nuchal line, reaches from the posterior border of the mastoid process to the occipital protuberance.

The Most Important Craniocerebral Determinations.—As it is now possible to localize lesions amenable to operation nearly everywhere within the cranium, and as almost all portions of the brain, except the mid regions of the base, are accessible to the surgeon, it may be necessary to indicate on the skull almost any fissure, convolution, or special part; but some fissures, convolutions, and regions are of paramount importance because of their functions, and of the frequency with which they are the seats of disease, and also because, in some instances, they serve as places of departure in mapping out the cranium. Among fissures which may need to be located are the Sylvian, central, precentral, retrocentral, first and second frontal, first and third temporal, intraparietal, parieto-occipital, and the transverse fissure between the occipital lobe and the cerebellum. Of these the most important are the Sylvian, the central, and the parieto-occipital. Among convolutions topographically important in craniocerebral work are the precentral and postcentral, the angular, the first, second, and third temporal, the second frontal, and the cuneus. It may also be necessary sometimes to define the position and extent of an entire lobe or a large part of it, as of the prefrontal, the lateral portion of the temporal, the occipital lobe, or a lateral lobe of the cerebellum. Numerous methods of determining craniocerebral lines and points are to be found in the textbooks. Some of these are simply slight modifications of others. All have their merits, and nearly all have some defects. Considerable experience in craniometrical measurements, as well as in the neurological aspects of cranial surgery, has convinced me that it is often difficult, if not impossible, to designate upon the scalp some of the well known points of the uncovered skull, such as the bregma, lambda, and parietal eminence. Those methods are therefore the best which select as points of departure landmarks which can always be determined.

Craniocerebral Variations.—The sutures even of the adult skull do not bear fixed relations to the fissures and convolutions. Considerable variations have been noticed. A comparison of Cunningham's illustrations with similar illustrations by others will show that the squamous suture is represented as above, below, and nearly on a line with the fissure of Sylvius; and other similar sutural variations are indicated. Anderson and Makins found that the summit of the parietal eminence had a range of variation in position half an inch in the vertical and an inch in the horizontal direction. The bregma and lambda have also a considerable range of variation in position from the glabella and inion. According to Foulhouze, in children up to the third or fourth year the Sylvian

Fig. 245.



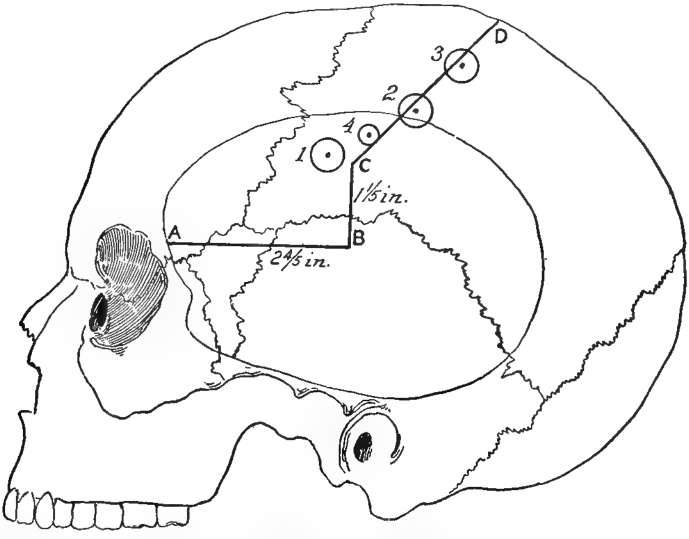
Craniocerebral guiding lines traced upon a photograph of one of the casts prepared by Professor Cunningham: *G*, glabella; *I*, inion; *M*, midsagittal point, midway between *G* and *I*; *A*, external angular point; *S*, squamosal point, intersection of oblique and frontal lines at junction of middle and lower thirds; *P*, parietal point, termination of oblique line equidistant with *b* from squamosal point; *E*, preauricular point; *a*, "commencement" of fissure of Sylvius, five twelfths of the distance from *A* to *S*; *b*, bifurcation of fissure of Sylvius, seven twelfths of the distance from *A* to *S*; *d*, termination of fissure of Sylvius, one half inch (1.25 cm.) above *P*, in a direction parallel to frontal line; *Ce*, central fissure; *C*, upper extremity of central fissure, three eighths of an inch (.9375 cm.) behind the midsagittal point; *c*, lower extremity of central fissure, carried to oblique line in direction of fissure, three eighths of an inch (.9375 cm.) in front of the squamosal point; *O*, parieto-occipital fissure, on sagittal line seven twelfths of the distance from *M* to *I*; the dotted line from *G* to *I* is the "sagittal line;" the line from *A* to *P* is the "oblique or squamosal line;" the line from *E* to *M* is the "frontal line." (After Anderson and Makins.)

fissure is more oblique and lies farther above the squamous suture. Dana found that in some young children the fissure lies just under the squamous suture, as in adults, but usually it is a little above it. In children the upper end of the central fissure is generally at or a little cephalad of the point given for adults.

Anderson and Makins's Craniometrical Lines.—Anderson and Makins suggest (1) a median *sagittal* line, from the glabella to

the inion ; (2) a *frontal* line, from the midsagittal point to the depression just in front of the ear at the level of the upper border of the meatus; (3) a *squamosal* line, from the most external point of the external angular process, at the level of the superior border of the orbit, to the junction of the middle and lower thirds of the frontal line, and prolonged for about an inch and a half behind the frontal line. The upper extremity of the central fissure was found by them to lie between the midsagittal point and a point three fourths of an inch behind it, and the lower extremity of this fissure they located near the squamosal line, about three fourths of an inch in front of its junction with the frontal line. The commencement of the lateral portion of the Sylvian fissure is not at a definite fixed point, but will usually be hit at a point from one and a half inches to two inches behind the angular process, the course of the horizontal portion of this fissure corresponding closely to the squamosal line.

FIG. 246.



Lucas-Championnière's method of determining the central fissure: AB, horizontal line two and four fifths inches long, drawn from the upper outer angle of the orbit; BC, perpendicular line one and one fifth inches long, to the position of the lower extremity of the central fissure; CD, course of central fissure from last point to half an inch behind the centre of the vertex; 1, speech centre (Broca's convolution); 2, 3, and 4 represent respectively the positions of the arm, leg, and face centres. (Lanphear.)

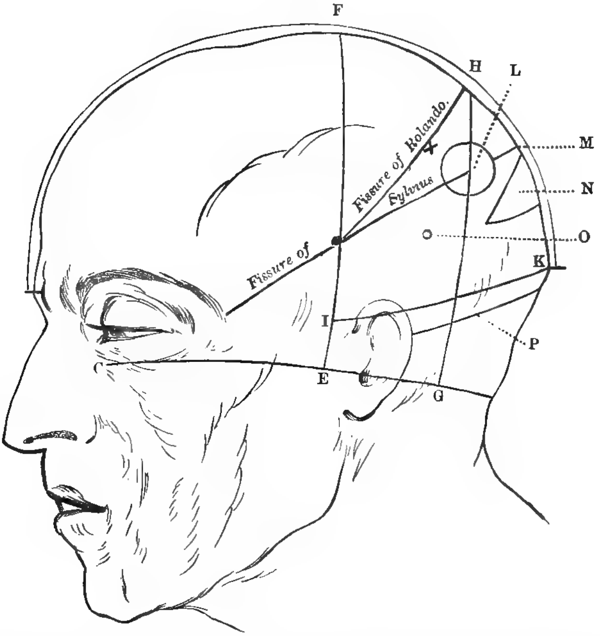
The parieto-occipital notch is placed at a point seven twelfths of the distance from the midsagittal point to the inion. The longitudinal sinus frequently deviates towards the right side in the caudal portion of its course. The points and lines given by Anderson and Makins are shown in Fig. 245.

Lucas-Championnière's Method of Determining the Central Fissure.—In Lucas-Championnière's method of determining the cen-

tral fissure, illustrated in Fig. 246, a line is drawn from the upper outer angle of the orbit, marked A, directly backward for a distance of two and four fifths inches (7 cm.) to the point marked B. From this point a perpendicular line is raised to C, and the line CD is drawn to a point half an inch (1.25 cm.) behind the centre of the vertex. This line represents the course of the central fissure within a fraction of an inch.

Reid's and Agnew's Landmarks.—Reid suggests to draw first a base line from the centre of the external auditory meatus to the inferior margin of the orbit. The longitudinal fissure is indicated by the line from the glabella to the inion. The transverse cerebellar

FIG. 247.



Agnew's diagram showing the cranial lines for cerebral localization: CG, Reid's base line drawn from the lower border of the orbit through the external auditory meatus to the base of the mastoid process; EF, anterior perpendicular line drawn from the sulcus in front of the ear to the top of the head; GH, posterior perpendicular line drawn from the posterior border of the mastoid process to the top of the head; ×, parietal eminence; O, position of postcornu, one inch and three quarters below the parietal eminence, and two inches and one quarter from the surface; IK, line drawn from the upper part of the occipital protuberance to the perpendicular EF, indicating the transverse cerebellar fissure; P, position of the lateral sinus; L, angular gyrus; M, parieto-occipital fissure; N, cuneus.

fissure is represented by a line drawn from the external occipital protuberance to the external auditory meatus. To determine the line of the fissure of Sylvius, a point is first taken one and one fourth inches (3.125 cm.) behind the external angular process of the frontal bone, and another three fourths of an inch (1.875 cm.) below

the most prominent part of the parietal eminence, the position of the fissure being indicated by a line drawn between these points. Two perpendiculars are drawn to the base line, one through the depression just in front of the ear, and the other through the posterior border of the mastoid process. These are extended to the longitudinal fissure, and a line is drawn from the junction of the most posterior of these perpendiculars with the longitudinal fissure to the junction of the most anterior with the Sylvian line. This represents the position of the central fissure. Agnew's method is a modification of that of Reid, and the lines and points used by Agnew are shown in Fig. 247 and described in its legend. The positions for trephining for the postcornu and the lateral sinus are shown on this figure. The Sylvian and central fissures are indicated by name. The base line, the perpendiculars, and the lines for the fissure of Rolando or central fissure are the same on Agnew's and Reid's diagrams. Various craniocerebral relations as given by Reid are shown in Fig. 248.

FIG. 248.

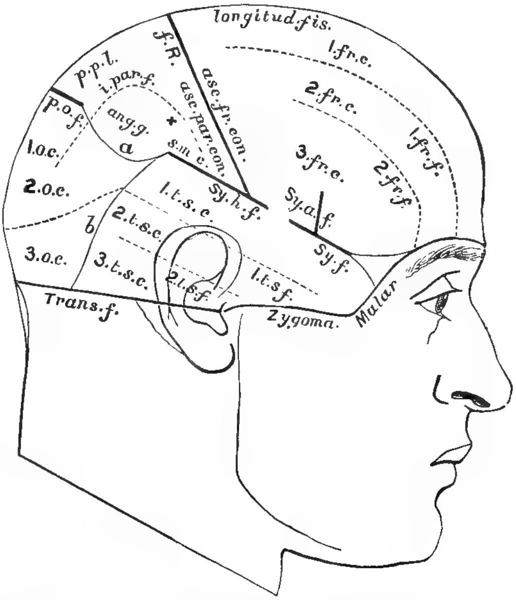
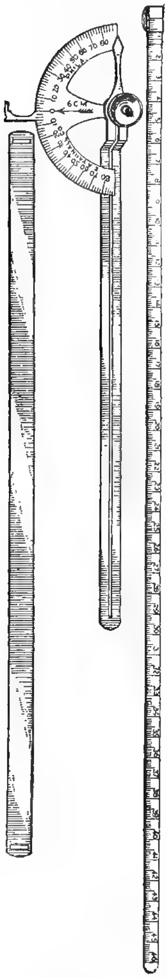


Diagram of craniocerebral relations: +, parietal eminence: *a*, convex line forming lower boundary of parietal lobe; *1.fr.c.*, first frontal or superfrontal convolution; *1.fr.f.*, first frontal or superfrontal fissure; *f.R.*, central fissure, fissure of Rolando; *Sy.f.*, fissure of Sylvius; *Sy.h.f.*, horizontal limb, and *Sy.a.f.*, ascending limb of Sylvian fissure; *p.o.f.*, parieto-occipital fissure; *i.par.f.*, intraparietal fissure; *ang.g.*, angular gyrus; *s.m.c.*, supramarginal convolution; *1t.s.c.*, supertemporal convolution; *1t.s.f.*, supertemporal fissure; *1o.c.*, first or superior occipital convolution; *p.p.l.*, superior parietal convolution (posteroparietal lobule). The convolutions and fissnres additional to those given in this legend are indicated on the figure by numbers. (Reid.)

Angle of the Central with the Longitudinal Fissure.—Thane determined an angle of sixty-seven degrees as that between the central and longitudinal fissures in front, and this angle has been generally accepted; but Horsley has shown that it varies somewhat with the shape of the head, believing that the higher the cephalic index the greater would be this angle, and *vice versa*. He assumed seventy-five as an average for the cephalic index as determined by

the calipers of Broca, and with this average he obtained for the central fissure an angle of sixty-nine instead of sixty-seven degrees. For every two degrees of variation of the cranial index he assumed one degree of variation of the central fissure: hence, if the cranial index be represented by seventy-seven instead of seventy-five, the angle for the fissure will be seventy instead of sixty-nine, and so on for other variations. He devised an instrument called the *fissure meter*, Fig. 249, which is provided with the means of rotating an arm representing the central fissure. It is shown applied to the head in Fig. 250. Hare estimated that the upper end of the fissure is at a point represented by fifty-five and seven tenths per cent. of the total distance of the glabella from the inion. Accepting the angle of sixty-seven, the fissure is supposed to extend laterally about three inches and three fourths (9.375 cm.), running from above downward and forward along this line. From these data Dr. Wilson, upon the suggestion of Professor Chiene, constructed an instrument called the *cyrtometer* (Fig. 251). One strip passes around the head through the glabella and the inion; another at right angles to this extends backward from the glabella to the inion. The latter strip is marked with two scales of numbers or letters, located at points accurately determined according to the proportion of fifty-five and seven tenths

FIG. 249.



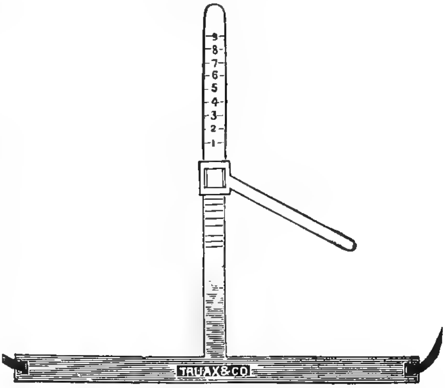
Horsley's meter for determining the position of the central or Rolandic fissure.

FIG. 250.

Horsley's fissure meter applied to the head.

to one hundred. When any given number or letter of one scale falls on the inion, the corresponding number or letter of the other indicates the upper limit of the fissure of Rolando. The course of the fissure is marked by a third narrow strip which is placed at an angle of sixty-seven degrees and is free to slide backward and forward on the longitudinal strip.

FIG. 251.



Wilson's cyrtometer.

Poirier's Nasolambdoidal Line.—Poirier has introduced a cranial line which will occasionally be found useful, and which he calls the *nasolambdoidal line.* It is drawn from the nasofrontal groove to a point two fifths of an inch (1 cm.) above the lambda. When the lambda cannot be felt, it can be found about two and four fifths inches (7 cm.) above the external occipital protuberance or inion. This line touches the lower pointed extremity of the pars triangularis of the subfrontal (third frontal) convolution, runs for a distance of from one and three fifths to two and two fifths inches (4 to 6 cm.) along the posterior limb of the fissure of Sylvius, grazes the inferior part of the supramarginal convolution, passes through the base of the angular gyrus, and abuts on the external parieto-occipital fissure. It passes through a point about one quarter inch (6 mm.) above the external auditory meatus. It will be seen that along this line are situated such important regions as Broca's convolution, the Sylvian fissure, the angular gyrus, and the point on the mesal edge of the hemisphere near which the cuneus would be reached. The angular convolution is situated on this line about two and four fifths inches (7 cm.) from the lambda.

Methods of Determining the Parieto-Occipital and other Fissures.—To outline the parieto-occipital fissure, the lambda is first found by measurement from the occipital protuberance, as indicated in the method given in the last paragraph, and a point three twenty-fifths of an inch (3 mm.) anterior to it is marked. The position of the fissure is indicated by a line drawn through this point at right angles to the longitudinal fissure, extending about one inch (2.5 cm.) on each side of the median line. To find the intraparietal fissure, the positions of the central fissure, fissure of Sylvius, parieto-occipital fissure, and parietal eminence are first determined. A point is next taken on a level with the bend of the fissure of Rolando and about four fifths of an inch (2 cm.) behind it. From this

a curved line is drawn upward and backward, keeping it half way between the central fissure and the parietal eminence as it ascends, and half way between the parietal eminence and the longitudinal fissure as it passes back. The line is continued until it reaches a point just outside of the external end of the parieto-occipital fissure. The precentral or vertical frontal fissure passes nearly vertically just posterior to the coronal suture. Its lower end is two fifths of an inch (1 cm.) above the Sylvian fissure, and one twelfth of an inch (2 mm.) behind the coronal suture. It lies from four fifths of an inch to one inch (2 to 2.5 cm.) anterior to the central fissure. The subfrontal fissure passes forward from the precentral fissure to a point a little above the superior stephanion, and continues forward in a line nearly identical with the frontal part of the temporal ridge. The superfrontal fissure begins at a point half way between the central fissure and a line prolonged up from the precentral fissure. This point should be from four fifths of an inch to one inch (2 to 2.5 cm.) in front of the central fissure. The fissure passes forward parallel to the longitudinal fissure, and its line if prolonged ends in the supraorbital notch. (Reid, Horsley, and Dana.)

Methods of Reaching the Cranial Fossæ.—Laplace has given particular attention to the methods of reaching and exploring fossæ at the base of the skull in intracranial operations. These methods are practically the same as those next to be described for exploring the orbital surface of the prefrontal lobe, the basal surface of the temporal lobe, and one lateral lobe of the cerebellum—in the last case extending the opening as low as possible. Laplace has suggested for the purpose of exploring the fossæ at the base of the brain, and for the removal of clots from these localities, the use of malleable wire instruments of different sizes, shaped like egg beaters: such an instrument was successfully used by him for the removal of a clot from the middle fossa.

Methods of Outlining the Prefrontal Lobe.—It may be necessary for certain purposes to reach the orbital as well as the lateral surface of the prefrontal lobe. In defining the cephalic or anterior extremity of this lobe, the varying thickness of the frontal portion of the cranium at this point, and also the line of the anterior fossa, must be taken into consideration. The thickness of the skull here ranges from one twelfth to one third of an inch (2 to 8 mm.), and the cephalic margin of the anterior fossa is about three-fourths of an inch (1.875 cm.) above the supraorbital arch. The fossa slopes gently backward, its posterior limit being the lower end of the coronal suture. In order to reach the orbital surface, therefore, it is necessary to trephine just above the supraorbital ridge, or in the temporal fossa along a line drawn from a point three fourths of an inch (1.875 cm.) above the supraorbital margin to the lower end of the coronal suture. The relations of the precentral, superfrontal,

and subfrontal fissures were given in the last paragraph, and, as these fissures traverse the prefrontal lobe, the points mentioned will serve as guides in reaching some parts of the lateral aspect of this lobe, which lies in front of the precentral fissure. To expose the subfrontal convolution, which on the left side is Broca's convolution, it is usual for surgeons to begin the incision through the temporal fossa at a point three fourths of an inch (1.875 cm.) caudad of the external angular process, and when the bone is exposed the

Fig. 252.

External lines and circles indicating the positions for applying the trephine in operations on the lateral sinus, the mastoid antrum, the cerebellum, and the temporal lobe: Reid's base line and perpendiculars are shown, the divisions on the lines indicating eighths of inches; A, position of opening for the lateral (sigmoid) sinus; B, position for anterior surface of the petrous bone, roof of the tympanum, and petrosquamous fissure, half an inch in diameter, its centre being situated seven eighths of an inch (2.1875 cm.) above the middle of the meatus; C, position for the mastoid antrum; D, position of opening for abscess of the temporal lobe; E, opening for cerebellar abscess; ×, × site of the tentorium in relation to the external boundary of the skull; the anterior × is the point where the tentorium leaves the side of the skull and is attached to the posterior border of the petrous bone. (Ballance.)

trephine is placed two inches behind this process. The lower, hinder part of the subfrontal convolution is closely related to the pterion, or junction of the great wing of the sphenoid with the frontal, parietal, and squamous bones,—which may serve as a guide in getting this position.

Methods of Outlining and Exposing the Temporal Lobe, Cerebellum, Mastoid Process, and Lateral Sinus.—As the neurologist and surgeon, particularly when intracranial abscess is under discussion, must consider diagnostic questions relating to the temporal lobe, cerebellum, mastoid process, and lateral sinus, the cranioencephalic relations of these parts will be described together in connection with the diagram Fig. 252. The temporal lobe is frequently the seat of abscess, and not infrequently of tumor or other lesion. The lower portion of the lateral aspect of the temporal lobe is indi-

cated by a line drawn from a point about half an inch (1.25 cm.) above the zygoma and the external auditory meatus through the asterion backward to the inion. The cephalic extremity of the lobe corresponds to the posterior border of the orbital process of the malar bone. At the external auditory meatus the temporal lobe is one and five eighths inches (4 cm.) wide. A trephine half an inch above the meatus would enter the lower portion of the lobe. The middle of the lobe is cut by a perpendicular passing along the posterior border of the mastoid. (Dana.) In Figure 252 at D is shown the usual position for the trephine opening for abscess of the temporal lobe, as given by Barker. The orifice should be half an inch in diameter (1.25 cm.), and one and a quarter inches (3.125 cm.) above the centre of the meatus. It may sometimes be desirable to reach the inferior or basal surface of the temporal lobe. The brain tumor which originated in the third temporal convolution, described, when speaking of the naming centre, on pages 345–347, might have been removed by operation if the diagnosis had been made sufficiently early. In order to reach this convolution it is necessary, as will be seen by examination of Fig. 244, to enter the skull considerably below the point marked D in Fig. 252. The operation for the removal of the Gasserian ganglion, which has been performed and described by Horsley, exposes this portion of the temporal lobe. It consists in turning down a large temporal flap and removing almost the entire squamous portion of the temporal bone. The lobe can be raised by a broad retractor and the exposed space illuminated by the electric light. In Fig. 252 is shown, at E, the position of the trephine opening designed to expose one lateral lobe of the cerebellum, which opening should be half an inch in diameter (1.25 cm.) and about one and a half inches (3.75 cm.) behind and one fourth of an inch (6.25 mm.) below the centre of the meatus. A trocar and canula directed forward, inward, and upward would strike an abscess of the interior portion of one of the lateral lobes of the cerebellum. The mastoid antrum is exposed by a trephine opening at C, Fig. 252, which is one fourth of an inch above (6.25 mm.) and half an inch (1.25 cm.) behind the centre of the meatus. The trephine should be directed inward and slightly forward and downward. In Fig. 252 are also shown the position and relations of the lateral sinus, the point A being the position of the trephine opening to enter it. The position, B, for reaching the anterior surface of the petrous bone, the roof of the tympanum, and the petrosquamous fissure, is also shown.

Tapping the Ventricles.—Keen has suggested tapping the ventricles as a systematic operation for dropsy, abscess, and perhaps other affections of the ventricles. He regards as most practicable three routes, for which he gives the following directions. (1) Trephine half way from the inion to the upper end of the central fissure,

one half to three quarters of an inch (1.25 to 1.875 cm.) to either side of the middle line. Puncture towards the inner end of the supraorbital ridge of the same side. The puncture will pass through the precuneus, and the normal ventricle will be struck at some point in the posterior horn at from two and a quarter to two and three fourths inches (5.625 to 6.875 cm.) from the surface of the scalp. (2) Trephine at one third of the distance from the glabella to the upper end of the central fissure, and one half to three quarters of an inch (1.2 to 1.875 cm.) to either side of the middle line. Puncture in the direction of the inion. The puncture will traverse the first frontal convolution well in front of the motor zone, and the normal ventricle will be struck in the anterior horn at about two to two and a quarter inches (5.0 to 5.625 cm.) from the scalp. (3) Trephine one and a quarter inches (3.125 cm.) behind the meatus, and one and a quarter inches (3.125 cm.) above Reid's base line, and puncture towards a point two and a half inches (6.25 cm.) directly above the opposite meatus. The puncture will traverse the second temporal convolution, and enter the normal lateral ventricle at the beginning or in the course of the descending cornu at a depth of about two to two and a quarter inches (5.0 to 5.625 cm.) from the surface. In the third route the measurements, which are those for an adult skull, should be somewhat reduced for children. All these measurements are to a normal ventricle. In a distended ventricle the distances would be proportionately less. Keen regards the lateral route as the best.

POSTMORTEM EXAMINATIONS OF THE BRAIN ESPECIALLY WITH REFERENCE TO THE LOCALIZATION OF LESIONS.

For full directions regarding the methods of postmortem examinations of the brain, manuals of autopsies and works on pathological anatomy must be consulted.* In medical centres autopsies are made by those especially trained for the work; but many important facts are lost to science through lack of knowledge of a few details such as will be given in this section.

Removal of the Brain from the Skull.—To prevent mutilation, the cut into the scalp is made from within outward, one of the best positions for the incision being from one mastoid to the other, over or just behind the vertex. The temporal muscles should be left in position, as subsequently a few stitches through them are of assistance in keeping the skull cap steady. The saw cut through the skull may be circular, as usually made, or may be wedge-shaped

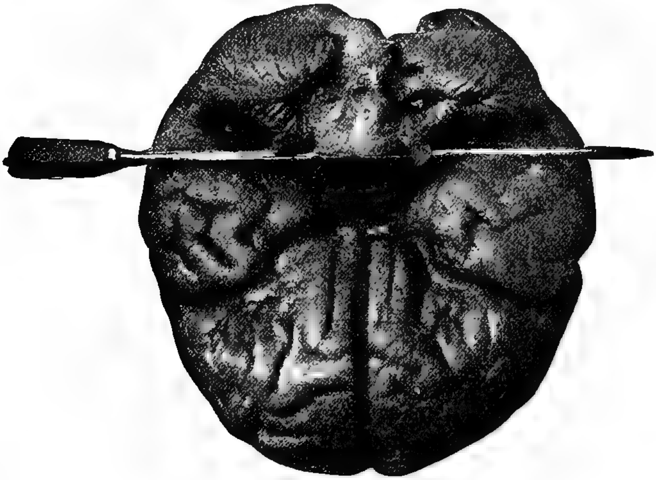
* Among useful manuals are those of Virchow, Bevan Lewis, Harris, Blackburn, Goodall, Hektoen, Hamilton, and Dejerine. The illustrations of brain sections in Fig. 257 and Fig. 258 have been redrawn, modified, and reduced in size, from plates in Blackburn's Manual of Autopsies. The illustrations on page 403 have been slightly modified from Hamilton's Pathology.

either in front or behind, so that after the autopsy the bones will fit together more neatly. Care should be taken to avoid injuring the meninges and brain with the saw or the chisel, and, if possible, the dura should be left intact. If the adhesions are strong, the membrane and the bone should be carefully separated with the scalpel and fingers, or the dura may be slit in the line of the saw cut and this portion of the membrane and the calvarium may be removed together. In an autopsy for medico-legal purposes it may sometimes be best not to use the chisel and mallet in opening the skull, as it might be asserted that a fracture which existed before death was thus produced, or that the brain and its membranes were in this way injured; but in such a case the sawing should be done with great care, as otherwise similar objections might hold against the use of the saw, since not infrequently the dura and even the arachnopia and brain are cut into in carelessly made autopsies. The inner aspect of the skull should be examined for caries, exostoses, old fractures, extravasations, or other abnormal conditions. The longitudinal sinus can be explored by opening it with the scalpel or bistoury before removal of the brain. The dura is severed from its connection with the crista galli by lifting it and cutting the falx in front; an incision is then made in this membrane in the line of the sawn bone as far backward as the tentorium, and the convexity of the dura, with the falx, is carefully detached and drawn backward. It may be advisable after removing the calvarium and the convex dura to examine the brain partially while in position, to do which its upper surface should be kept as nearly level as possible, and some of the incisions to be hereafter described used to open the ventricles. A syringe with a long nozzle can be employed to withdraw the ventricular fluid. In the process of removal of the brain from the skull—which is the next step—the brain should be thoroughly supported on all sides. Its frontal lobes are tilted a little backward, and the nerves and vessels first met with are carefully severed. When the tentorium is reached it is incised on each side along the lateral sinus. Still gently tilting the brain backward, the remaining nerves and vessels are divided, and finally, by passing a knife as low down as possible, the spinal cord is cut across and the brain is withdrawn from the skull.

Inspection of the Brain and the Cranial Cavity.—After removal of the brain the investigation of the dura should be carefully completed, and both surfaces of the pia should be examined. The condition of the vessels both as to their walls and as to their contents should be noted. An examination of the edges of the sawn bones and of the inner surface of the floor of the skull should never be neglected; and it may be important to examine the Gasserian ganglion and the portions of the cranial nerves remaining behind in the grooves, cavities, and foramens of the skull.

Method of making Autopsies to save the Brain for Subsequent Examination.—In many cases the full examination of a brain need not be made at the time of the autopsy. Valuable material is ruined by improper cutting of fresh specimens. When the knife is passed through a fresh brain or cord, considerable tissue on both sides of the cut may subsequently in the process of hardening be found to be destroyed. The method recommended by Dejerine probably causes the least destruction of material. After the brain has been removed, it should be placed resting on its upper surface, and by means of a long, thin, and sharp knife the pons and preoblongata should be cut through transversely just above the exit of the fifth nerves, as shown in Fig. 253, thus separating the cerebellum with most of the pons and oblongata from the rest of the brain. In the older methods commonly employed, transverse cuts are made through

FIG. 253.

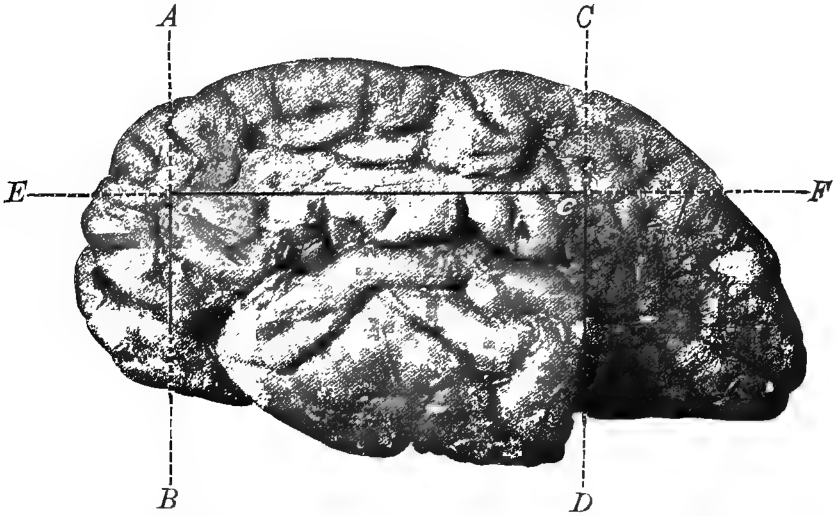


Incision through the pons (just above the exits of the roots of the fifth nerve) in Dejerine's method of making an autopsy.

the crura, but these sections always greatly injure important structures (such as the nuclei of the third nerves), especially if it is intended to make subsequently a thorough microscopical examination. After the incision through the pons has been made, the occipital and frontal lobes should be removed by vertical cuts in the positions of the lines *CD* and *AB* at the two extremities of the callosum (Fig. 254). A horizontal cut is next made just above the superior surface of the caudatum along the line *ac*. These are the only incisions absolutely necessary at the time of making the autopsy, but after making these, if it is thought advisable, the portions of the two cerebral hemispheres still attached to each other may be sepa-

rated by a cut through the callosum and the exact centre of the space between the cerebral peduncles, unless for special reasons it is thought

Fig. 254.



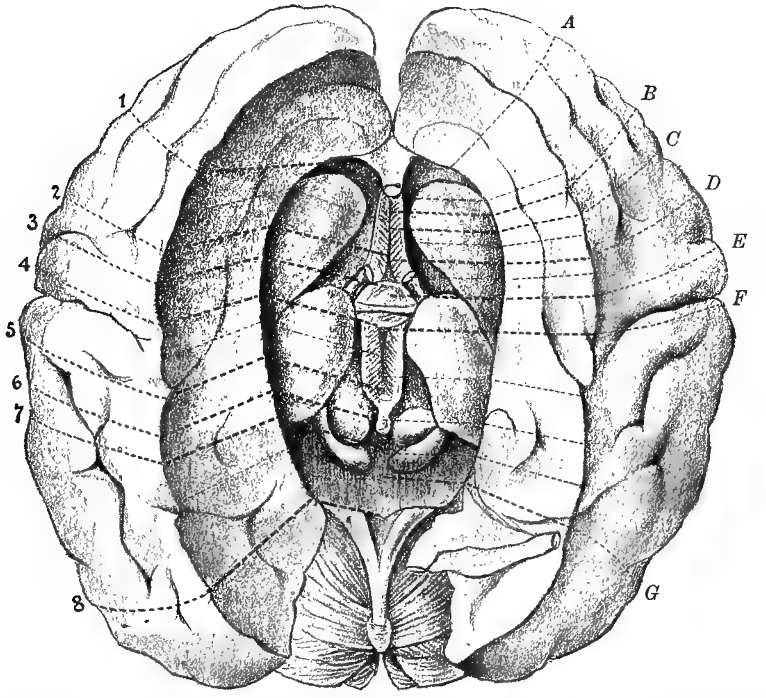
Lines of incision into the brain when an autopsy is made with the view of completing the examination after hardening of the specimen: *AB* and *CD*, lines showing the position of cuts for the removal of the occipital and frontal lobes; *ac*, line of horizontal incision, just above the superior surface of the caudatum.

better to make the incision a little to one side. If a week or two be allowed to pass before making this latter incision, the tissue will be less likely to be injured at the point of section. If the lesion is in the frontal lobe, the cut *AB* should not be made, and the cut *ca* may be extended forward to *E*. In the absence of indications of lesions of the occipital lobe, the line *Eac* may be extended to *F*. No other cuts should be made. If a lesion is not found, and yet there is good reason from the clinical history to believe that one exists, the brain should be hardened and cut with the microtome. Each cerebellar lobe should be cut away anteroposteriorly, close to the pons, as otherwise the cerebellum may not harden properly.

Methods of entering the Brain and making Transections of the Cerebrum.—Instead of following the method of Dejerine just described, in which the chief examination of the brain is made after hardening, it may be necessary or desirable to make a more complete examination at once. One of the best known procedures is to enter the lateral ventricles by a perpendicular incision into the callosum on each side of its raphe. The precornu and postcornu are next exposed by further incisions. A small sharp knife is then introduced into the foramen of Monro and a cut is made upward and forward through the callosum and subjacent structures, the portions

anterior and posterior to the section being turned forward and backward respectively. The ventricles having been entered, incisions into the hemispheres can be made, care being taken not to destroy the central parts of the base, chiasm, fornix, callosum, etc. When the callosum and fornix are turned backward, the velum interpositum comes into view. An incision can be made through the callosum and posterior pillars of the fornix on one side, and these parts

FIG. 255.



Method of making transections from within outward of the ganglia, capsules, and hemispheres. The heavy oblique lines (1, 2, 3, 4, 5, 6, 7, and 8) indicate the direction and approximate positions of the Pitres-Blackburn sections (Figs. 257 and 258); the lines A, B, C, D, E, F, and G, on the other side of the brain, at right angles to the brain axis, indicate approximately the position of Hamilton's sections (Fig. 259); the lighter intermediate lines indicate the position of other sections, any number of which can be made. The lines are represented as reaching the median line, but the incisions begin in the ventricles at the inner margins of the bodies cut. The quadrigeminal body is not included in the sections.

reflected to the other side. Various methods of making sections of the cerebrum either before or after opening the ventricles have been recommended. A method commonly employed, leaving the hemispheres in position after opening the ventricles, is to make transverse incisions from within outward through the ganglia and related portions of the hemicerebrums, making similar transections into the occipital and frontal lobes. Two methods of doing this are

shown in Fig. 255,* the incisions on one side being parallel with the central fissure (Pitres-Blackburn method) and on the other at right angles to the long axis of the brain (Hamilton method). Virchow's method was to divide the ganglia by fan-shaped radial incisions starting at the peduncles of the cerebrum. Pitres first described a series of six now well known verticotransverse sections: the first through the prefrontal region (prefrontal section); the second through the base of the three frontal convolutions (pediculofrontal section); the third through the precentral convolution (frontal section); the fourth through the postcentral convolution (parietal section); the fifth through the inferior and superior parietal convolutions (pediculoparietal section); and the sixth through the occipital lobe (occipital section). Blackburn has suggested two additional sections, which he designates the anterocentral and posterocentral, the former passing through the precentral and the latter through the retrocentral fissure. The lines along which these sections are made are shown in Fig. 256, and the appearances presented by them in Figs. 257, 258, and 259. Of course, in cutting from within, the convolutions cannot so readily be used as guides, but sections along the heavy lines on the left side of Fig. 255 will be nearly correct. Instead of verticotransverse, verticolongitudinal sections may be employed, long, smooth incisions deep enough to expose all parts to within a few lines of the convolutions of the base being carried through both the thalamus and the striatum and out into the hemispheres, a few additional longitudinal sections being made in the white matter. The sections of Pitres, either as originally given or as modified by Nothnagel or Blackburn, have proved of great value to anatomists and pathologists, but it is necessary, as noted by Hamilton, to bear in mind certain sources of error when these sections are used. Pitres's sections are intended to be made parallel with the central fissure, but the parts cut may differ considerably according to the position and condition of the brain. The appear-

FIG. 256.

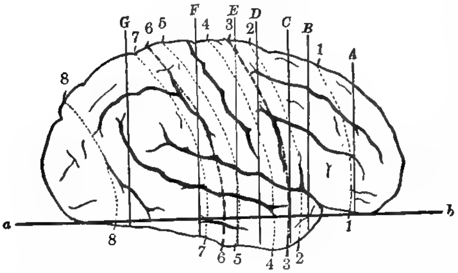


Diagram showing the lines of the Pitres-Blackburn and Hamilton transections: *ab*, horizontal line from frontal to occipital lobe; the numbers from 1 to 8 indicate the positions of the former, and the letters from *A* to *G* those of the latter. The appearances presented by transections through the numbered lines are shown in Figs. 257 and 258, and those presented by transections through the lettered lines in Fig. 259.

* Of course the appearances shown in the sections of Pitres, Blackburn, and Hamilton are present only when a method is followed which keeps together the basal ganglia and the cortex.

ances properly attributable to such sections are such as are seen after incising the brain while still in position in the skull. When the brain is removed from the skull and is placed upon a plate or a board, it may flatten out considerably; and therefore cuts made

Fig. 257.

Transections of the brain according to Pitres and Blackburn: the numbers 1, 2, 3, and 4 indicate sections corresponding to lines with the same numbers in Fig. 256: 1, prefrontal section; 2, pediculofrontal section; 3, anterocentral section of Blackburn (through the precentral fissure); 4, frontal section (through the precentral convolution); *A*, superfrontal, *B*, medifrontal, *C*, subfrontal, *D*, orbital convolutions; *E*, callosum; *F*, caudatum; *G*, internal capsule; *H*, lenticula; *I*, insula; *K*, precentral, *L*, postcentral, *M*, supertemporal, *N*, meditemporal, *O*, subtemporal convolutions; *P*, thalamus; *Q*, external capsule; *R*, claustrum; *T*, temporal lobe; *U*, superior parietal lobule; *V*, inferior parietal lobule; *W*, occipital lobe; *X*, extreme capsule; *cc*, central fissure.

into brains taken from the skull may not strike the same places for different brains, nor the same as in brains examined in the skull. The planes of the sections will differ somewhat according as the brain is rested on its convexity or on its base, and according to its size or even the sizes of the convolutions. Hamilton recommends

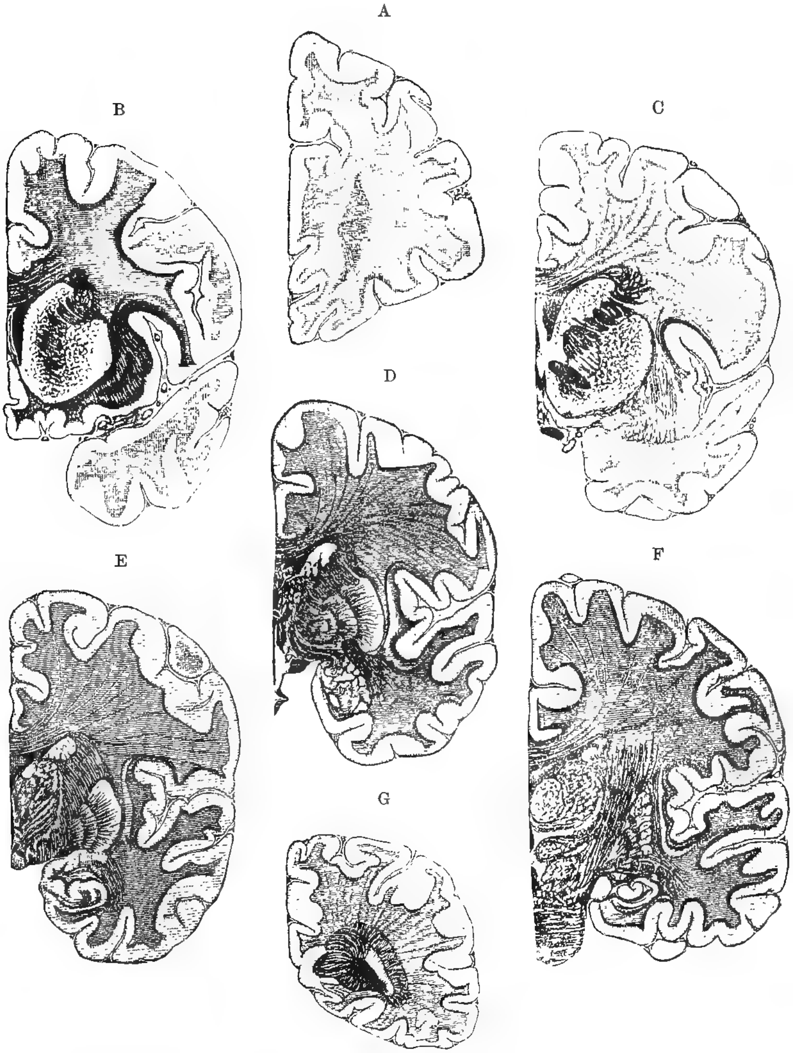
that the brain when removed from the skull should be placed in one constant position,—vertex downward on a board, with the tips of the occipital and frontal regions in one horizontal line. For ordinary purposes, having first removed the cerebellum, pons, and oblongata, he recommends that the brain be sliced through in different locations as indicated by letters on Fig. 256 : (*A*) through the cephalic

FIG. 258.

Transections of the brain according to Pitres and Blackburn : the numbers 5, 6, 7, and 8 indicate sections corresponding to lines with the same numbers in Fig. 256 : 5, parietal section (through the postcentral fissure) ; 6, posterocentral section of Blackburn (through the retrocentral convolution) ; 7, pediculoparietal section ; 8, occipital section ; *E*, callosum ; *F*, caudatum ; *G*, internal capsule ; *H*, lenticula ; *I*, insula ; *L*, postcentral ; *P*, thalamus ; *Q*, external capsule ; *R*, claustrum ; *S*, hippocampus ; *T*, temporal lobe ; *U*, superior parietal lobule ; *V*, inferior parietal lobule ; *W*, occipital lobes.

half of the subfrontal convolution ; (*B*) through the tip of the temporal lobe and operculum ; (*C*) just cephalad of the chiasm ; (*D*) through the infundibulum, passing obliquely through the temporal, precentral, and superfrontal convolutions ; (*E*) through the albicantia ; (*F*) through the cephalic margin of the pons ; and (*G*) across the front of the angular gyrus. The appearances of the sections

Fig. 259.



Appearances presented by Hamilton's transections of a hardened brain. The sections A, B, C, D, E, F, G, correspond to the lettered lines in Fig. 256.

thus made on one side of the brain are shown in Fig. 259, the letters corresponding to the section lines given above.* These sections are

* The figures were drawn by Hamilton from sections of preparations made by his gelatin potash method, and were traced by him on gelatinized glass from the preparations. In this method the specimen is hardened in Müller's fluid, then frozen sections are cut, and these by a special process are encased in gelatin and later immersed in liquor potassæ, then stretched in Müller's fluid and mounted. The details of this valuable process, which is somewhat complicated, are given in Hamilton's Pathology.

presumably at right angles to the anteroposterior axis of the brain, and hence are not parallel with the central fissure, and they will therefore cut various convolutions more or less obliquely. The ganglia, tracts, and other structures in the interior of the brain in these sections hold relations to the convolutions which in some instances differ greatly from those observed in the Pitres-Blackburn sections. It is sometimes absolutely necessary to examine the brain in the fresh state, as when in private practice it may not be possible to remove the organ for subsequent investigations, or when it is important for medico-legal reasons to describe appearances as they are when first seen. Certain advantages are secured by hardening the brain before making sections: all sections can be kept as permanent preparations; the preparations are equally useful for examination with the naked eye or with the microscope; and any lesion causing softening is more distinctly marked than in the fresh state, on account of the softened tissue being fixed. (Hamilton.)

The Interior Structures of the Cerebrum projected on its Convex Surface.—The diagram Fig. 260, redrawn from one prepared by Stacey Wilson, will prove useful in autopsies and in cerebrotopographical work. It shows the relations of the internal capsule and other structures of the interior of the cerebrum to the convolutions. Their outlines are in the illustration projected vertically upon the surface. The main portion of the internal capsule, which lies between the upper part of the lenticula and the caudatum, is shaded with fairly close lines. The outer boundary of the capsule is, of course, an arbitrary line. The line taken was the highest point where the capsule touched the lenticula,—that is, the upper and internal edge of this nucleus as seen in transverse section. The prolongation of the capsule inward to form its genu, and its continuation into the crusta of the crus, are shaded faintly. These two tracts are shown passing into the pons and finally under the cerebellum.

Examinations of the Cerebellum, Pons, and Oblongata.—To examine the cerebellum, pons, and oblongata, either when they are still united with the rest of the brain or after they have been separated by severing the crura, an incision should be carried from above downward through the middle of the vermis, care being taken not to wound the floor of the fourth ventricle. If it is desired to preserve the vermis entire, this section can be made through its line of junction with either lateral lobe. After the perpendicular section, further incisions can be made outward in the direction of the medipeduncles, or the cuts for special reasons may be longitudinal or transverse. The cerebellum can be examined alone by first carefully separating its under surface from the oblongata and pons and then cutting across its peduncles one after the other. Internally it is necessary to localize lesions of the cerebellum with reference to its central stem of white matter, its gray cortical laminæ, the den-

tatum, and other deposits of gray matter. The floor of the fourth ventricle should be minutely explored, for here may be found most interesting lesions, both primary and secondary. Descriptions of lesions should be definite and accurate, and they can easily be made so by taking as points of departure such readily recognizable land-

FIG. 260.

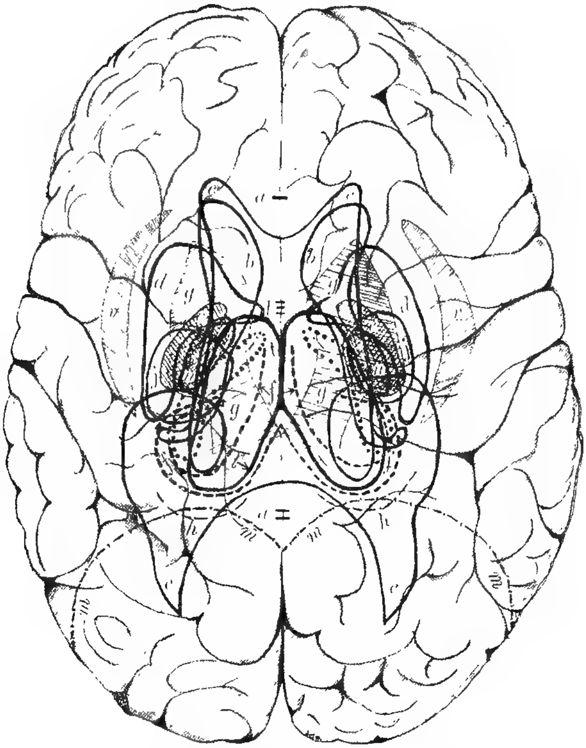


Diagram showing the relations of the internal capsule and other structures of the interior of the cerebrum to the convolutions of its convex surface: *a*, *a*, limits of the callosum; *b*, *b*, caudatum; *c*, *c*, *c*, *c*, outline of the lateral ventricles; *d*, *d*, lenticula; *e*, *e*, and *n*, *n*, two divisions of the putamen of the lenticula; *f*, *f*, fornix (only its posterior portion and the descending pillars are shown); *g*, *g*, internal capsule (its outer and upper part between the lenticula and the caudatum is shaded darkly, its lower portion and the crusta are shaded faintly); *h*, *h*, marks the situation for a short distance of the tenia; *i*. *i*, pregeniculum and optic tract; *j*, *j*, gray nucleus below the lenticula and anterior to the pes hippocampi; *l*, precommissure, marked only in the middle line; *m*, *m*, *m*, *m*, outline of cerebellum; *r*, *r*, outline of insula. (After Stacey Wilson.)

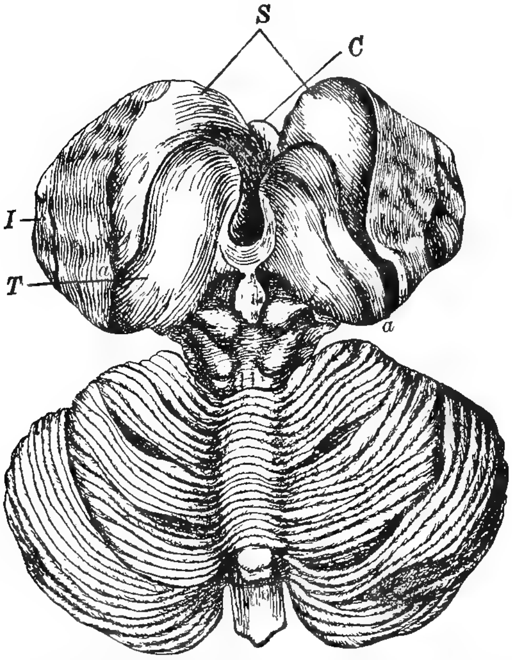
marks as the median furrow, the medullary striæ, the locus ceruleus, and the ala cinerea.

Methods of Separating the Brain into its Component Parts.—As not only the weight of the brain as a whole, but also the weight of its component parts, may be a matter of clinical and pathological interest, and as for other reasons it may be desirable to subdivide

the brain upon some regular plan, it is necessary to have convenient methods of making this dissection. Meynert's method of separating the brain into the brain mantle, brain stem, and cerebellum is that which has been most commonly employed. By Meynert, and by anatomists and neurologists in general, the term *pallium*, or *brain mantle*, has been made to include all portions of the cerebral cortex, as usually recognized and described, and the *brain stem*, or *caudex*, the rest of the encephalon, with the exception of the cerebellum,—namely, the striatum, thalamus, quadrigeminal body, pons, oblongata, and their appendages and connections. In Meynert's method the brain is placed with its base upward, the pia being left in position. The fissure of Sylvius is opened to its fullest extent, so as entirely to expose the insula. The prefrontal lobes are next separated from the striate bodies, to do which the two frontal lobes are pulled gently apart until the knee of the callosum is uncovered, when a cut is made first on one side and then on the other from the depression just in front of the anterior perforated space, in a nearly horizontal direction, to a point about four tenths of an inch (1 cm.) behind the knee of the callosum, where the knife is turned vertically and passed into the precornu. The incision is continued along the anterior furrow of the insula. The oblongata, pons, and cerebellum are now lifted, and the pia is detached laterally and behind at the opening of the great transverse fissure, bringing into view the third ventricle and the descending horns. Lifting the temporal lobe by its apex and holding the knife in a nearly horizontal position, the junction of this lobe with the base of the brain is cut through until the cephalic extremity of the descending horn is entered, the same procedure being repeated on the other side. Another cut is made from the descending horn as far backward as the posterior angle of the insula. Again elevating the mass (the cerebellum, pons, and oblongata), an incision is now carried from the extremity of the last cut (at the posterior angle of the insula) along the outer limiting furrow of the insula until it meets the cut first made. Elevating still further the cerebellum and brain stem, the anterior pillars of the fornix and the septum lucidum come into view and are severed, and the dissection is completed. In pursuing this method the two subdivisions of the striatum and the insula are regarded as parts of the brain stem; but, as already stated, the caudatum is undoubtedly a cortical formation, and it is probable that the lenticula also belongs to the cortex in part or altogether. The brain stem proper does not, therefore, extend cephalad of the thalamus. The brain mantle includes all parts of the cerebrum derived from the first secondary vesicle or prosencephalon, while the brain stem is constituted of all the parts, except the cerebellum, derived from the other cerebral vesicles. It is practically impossible to separate the thalamus neatly and quickly from the striate bodies in an ordinary autopsy, but by a deviation from Mey-

nert's method a dissection can be made which will more nearly detach the true brain stem from the brain mantle. The brain being with its convexity downward, lifting the oblongata, pons, and cerebellum, and opening the great transverse fissure and middle horns as in Meynert's method, an incision having a somewhat backward inclination is made on each side as nearly as possible between the anterior and lateral boundary of the thalamus and the caudate body. The incision can be so directed as to leave only a small portion of the

FIG. 261.



Brain stem and cerebellum separated from the brain mantle by Meynert's method : *S*, striatum ; *C*, callosum ; *I*, insula ; *T*, thalamus. An incision through the heavy line *aa* inclined somewhat backward, and carried through to the base, would separate the thalamus from the parts anterior to it, leaving the true brain stem. (Hektoen, after Siemerling.)

posterior parts of the lenticula and caudatum attached to the brain stem, from which they can be dissected, or a slight allowance can be made for their weight. The line *aa* in Fig. 261 indicates the general course of this incision.

The Separation and Weighing of Cerebral Lobes.—Sir James Crichton-Browne, in a series of investigations on the weight of the brain and its component parts in the insane, obtained data with reference to the weight of the whole brain, of each hemisphere, of the cerebellum, of the pons, and of the oblongata. His method is described as follows. Laying the brain in a small trough, a few rents are made in the pia and a few incisions into the callosum, so as to

drain away the serous fluid present. After weighing it as a whole, the brain is placed with its upper surface downward upon a board, the cerebellum is raised in the left hand of the operator, and two clean cuts are made with a large brain knife between the crura and the pons. The hemicerebrums are turned over and separated from each other by one long sweep of the knife in the central line of the callosum. The cerebellum is separated from the pons by incisions through its peduncles, and the pons is severed from the oblongata by a cut through the furrow between the ventral surfaces of these two parts. Each portion of the brain is then separately weighed. The cerebral hemispheres can also be subdivided into anatomical or physiological lobes, or into other parts, according to the inclination or intention of the investigator, and these portions can be separately weighed.

Brain Weight.—The weight of the brain in a man is about two per cent. of the weight of his entire body; the relative weight of a woman's brain to the body is nearly the same. The relative weights of different parts of the brain are—for the frontal lobes, twenty-eight per cent.; for the parietal lobes, thirty-six per cent.; for the occipital lobes, ten per cent.; for the temporal lobes, thirteen per cent.; for the insula, nine per cent.; and for the pons, one and a half per cent. The weight of the cerebellum is about one eighth that of the cerebrum. (Dana.) Many valuable statistics have been collected as to the weights of male and female brains, of the brain at different ages, of the brains of the insane and the idiotic, and of the brain of man as compared with the brains of other animals. As the result of extensive comparisons by numerous investigators, it has been found that in a large proportion of cases the weight of the adult human brain ranges for the male between forty-six and fifty-three ounces (1303 and 1502 grammes), and for the female between forty and forty-seven ounces (1133 and 1332 grammes). Boyd found that the mean weight of the male brain at from twenty to forty years of age was forty-eight ounces (1360 grammes), and that of the female brain forty-three and one half ounces (1230 grammes). (Quain's Anatomy.) According to some authorities, the brain attains its maximum weight between the ages of fourteen and twenty years; according to others, between the ages of twenty-five and thirty-five in the male and a little earlier in the female. It attains about five sixths of its ultimate weight by the end of the seventh year in the male, and ten elevenths in the female. Between forty and fifty years a slight diminution in weight occurs, and a still greater decrease takes place in the fifth decade, while from sixty years onward the rate of decrease is progressively more rapid. The general law of decrease, however, has many exceptions. The principal conditions that modify brain weight are age, sex, body weight, stature, mental power, and degree of education.

Brain Weights at Different Ages.—The following table from Quain's Anatomy was compiled from observations by Boyd:

	Males.		Females.	
	Ounces.	Grammes.	Ounces.	Grammes.
Children stillborn at term	13.1	393	12.2	347
Children alive at term	11.1	330	9.9	283
Under 3 months	17.1	493	15.9	451
From 3 to 6 months	21.2	602	17.4	560
From 6 to 12 months	27.3	776	25.3	727
From 1 to 2 years	32.5	941	29.7	843
From 2 to 4 years	38.3	1,095	34.9	990
From 4 to 7 years	40.1	1,138	47.1	1,335
From 7 to 14 years	45.9	1,301	40.4	1,154
From 14 to 20 years	48.4	1,374	43.7	1,244
From 20 to 30 years	47.0	1,333	43.6	1,237
From 30 to 40 years	48.1	1,364	43.0	1,220
From 40 to 50 years	47.6	1,351	42.7	1,212
From 50 to 60 years	47.3	1,343	43.0	1,220
From 60 to 70 years	46.3	1,313	42.6	1,208
From 70 to 80 years	45.4	1,288	41.0	1,168
Over 80 years	45.3	1,283	39.6	1,125

Brain Weight in Relation to Body Weight, Stature, and Mental Condition.—A definite relation seems to exist between brain weight and body weight and stature. Quain gives 1 to 41, in healthy individuals dying suddenly, as the ratio between the weight of the brain and that of the body, but the relative weight is liable to great variation. The influence of stature on brain weight has been investigated by Marshall. Brain weight increases with the increase of body stature, but taller persons have relatively less brain matter than shorter ones, although absolutely they have more. The proportion remains larger in the male. Marshall came to the conclusion that although woman has less cerebral matter than man when judged by her height alone, and more when only her body weight is considered, yet when both sexes are judged by an assumed common measure having reference to both factors their cerebral endowment is seen to be practically equal. The following table is compiled from data given by Davis, Simms, Wilson, and Bastian. The small brain weight of the Hindoos is probably related to their prevailing small stature; and among the Europeans the Latin races have slightly less brain weight than the Teutonic and Sclavonic races. According to Welcker, the average brain weight of Europeans is forty-nine ounces (1390 grammes). Flint, quoted by Simms, gives fifty and two tenths ounces (1422 grammes) as the average weight of a number of brains examined in New York. This is about the average weight given by Peacock for Scotch brains. Many of the estimates in the table have been obtained from measurements of the cubic contents of the skull cavity.

Race.	No.	Capacity.	Brain Weight.	
		Cu. In.	Ounces.	Grammes.
Australians	24	81.9	41.81	1185
Mexicans	25	81.7	41.71	1182
Hindoos	35	82.5	42.11	1193
Negro tribes	69	85.2	43.47	1232
Negroes	16	86.4	44.08	1249
Negroes in the United States	139	91.9	46.90	1329
Asiatics	210	87.1	44.44	1259
Javans	30	87.5	44.66	1266
Oceanic tribes	305	89.4	45.63	1293
Hawaiians	121	90.1	45.95	1303
North American Indians	18	90.6	46.23	1310
South American Indians	12	88.0	44.92	1273
Esquimaux	13	91.2	46.56	1319
Europeans	299	92.3	47.12	1335
English	21	93.1	47.50	1346

The weight of the brain varies with different forms of insanity. The following table is by Crochley Clapham :

Average Brain Weight in Different Forms of Insanity.

No.	Form of Insanity.	Male.	Female.	Mean of Sexes.
		Grammes.	Grammes.	Grammes.
19	Idiocy	1200.7	1077.7	1139.2
11	Imbecility	1331.2	1246.6	1288.9
254	Dementia (simple)	1356.8	1234.1	1295.4
209	Dementia (senile)	1348.5	1204.9	1276.7
59	Dementia (organic)	1347.7	1198.0	1272.8
235	Acute insanity	1441.6	1278.0	1359.8
243	General paralysis	1302.0	1134.3	1218.1
117	Epilepsy	1391.6	1217.6	1304.6
112	Chronic mania	1392.9	1268.2	1330.5
59	Brain wasting	1251.2	1195.2	1223.2

"The above shows that the highest average brain in both males and females is found in the acute forms of insanity, while the lowest average is, as might be expected, found in idiocy. Epileptic insanity, in both sexes, has a high average brain weight; chronic mania one still higher. In imbecility, the male average is below the general male average, while that for the females is higher than the general average for that sex. In simple dementia, both males and females have a larger brain average than the general average for their sex respectively. Senile and organic dementia, general paralysis, and cases of brain wasting have, in both males and females, an average less than the general average for the sex in the insane." (Crochley Clapham.)

Recorded Brain Weights of Eminent Men.—The following table of brain weights of eminent men has been compiled from numerous sources :

Name.	Occupation.	Age.	Ounces.	Grammes.
Abercrombie	Physician	64	63.0	1785
Agassiz	Naturalist	66	52.7	1495
Bennett (Hughes)	Physician	63	47.0	1332
Bischoff (Ch.)	Physician	79	51.2	1452
Broca	Anthropologist	56	52.3	1484
Byron	Poet	36	63.7	1807
Campbell (Lord)	Jurist	80	53.5	1516
Chalmers	Preacher	67	53.0	1502
Cuvier	Naturalist	63	65.6	1861
Dante	Poet	56	50.0	1420
De Morgan	Mathematician	73	52.7	1496
De Morny	Statesman	50	53.6	1520
Dirichlet	Mathematician	54	53.6	1520
Döllinger	Physiologist	71	42.6	1207
Dupuytren	Surgeon	58	50.7	1437
Fallmerayer	Historian	74	47.5	1349
Fuchs	Physician	52	52.9	1499
Gambetta	Statesman	44	40.8	1160
Gauss	Mathematician	78	52.6	1492
Goodsir	Anatomist	53	57.5	1630
Grant (Prof. R. E.)	Anatomist	80	45.5	1290
Grote	Historian	75	49.7	1410
Harless	Physiologist	40	43.6	1238
Hausmann	Mineralogist	77	43.2	1226
Hermann	Philologist	51	47.9	1358
Hermann	Politician	73	56.0	1590
Huber (J. N.)	Philosopher	49	49.7	1409
Kant	Philosopher	82	56.4	1600
Leidy	Anatomist	68	49.9	1415
Liebig	Chemist	70	47.6	1352
Meyr (Melchior)	Poet	61	49.9	1415
Pfeufer	Physician	60	52.4	1488
Schiller	Poet	46	63.0	1785
Simpson (James)	Physician	59	54.0	1533
Spurzheim	Physician	56	55.1	1559
Thackeray	Novelist	52	58.5	1658
Tiedemann	Physiologist	80	44.2	1254
Turgénieff	Novelist	74	74.7	2120
Webster (Daniel)	Statesman	70	53.5	1516
Whewell	Philosopher	71	49.0	1390
Whitman	Poet	73	43.3	1230
Wright (Chauncey)	Physicist	45	53.5	1516

In this connection it is interesting to note that unintellectual individuals frequently have high brain weights. One of the heaviest brain weights on record is that of a bricklayer who could neither read nor write, the weight of whose brain as recorded by Dr. James Morris was sixty-seven ounces (1899.45 grammes). Apparently there is no invariable nor necessary relation between mere brain weight and the degree of individual intelligence ; but high brain weights occur in larger proportions among civilized than among uncivilized races.

Determination of the Volume of the Brain.—A method of determining the volume of the brain is to take a vessel of convenient size and shape, with a capacious spout placed at an acute angle with its sides. Water is poured into this vessel up to the level of the spout. Fluid contained in the ventricles of the brain and the sub-arachnoid spaces is allowed to escape by several long incisions, and then the brain is gradually immersed, the displaced water as it escapes from the spout being caught and measured.

Determination of Cranial Capacity.—Cranial capacity may be determined by one of several methods. The foramina at the base of the brain are plugged with tenacious clay ; that used for modelling answers best. A small triangular piece is removed from the frontal bone ; the calvarium is readjusted at the base, the dura being left attached. The space left by the saw in removing the calvarium is filled with clay, and a narrow bandage with clay spread upon it is made to surround the cranium three or four times, covering this space. Sixty fluidounces of water having been measured, a sufficient quantity to fill the cranial cavity is poured into it by means of a funnel through the orifice in the frontal bone. The fluid which remains after having filled the cranial cavity is measured, and being deducted from the sixty ounces gives the amount employed, to which must be added half an ounce for the space occupied by the luting. The paraffin method is first to fill the foramina at the base with modeller's clay, as above described. After the removal of the calvarium, a triangular or wedge-shaped piece is sawed out of the occipital bone, but retained *in situ*. Having previously trephined a piece out of the frontal bone, the skull cap is replaced and retained by luting, as in the previous method. Through the orifice in the frontal bone, more of the paraffin is poured in until the cranial cavity is filled. When cool and solid, the calvarium as well as the wedge-shaped piece of bone from the occiput is removed, and then gentle pressure from behind tilts the solid mass out of the cranium, when it will be found to form an exquisite mold of the interior. The mold thus obtained is to be measured by displacement, which gives the cranial capacity. (W. Bevan Lewis.)

Specific Gravity of the Brain.—A method of determining the specific gravity of the brain is to immerse the brain or portions of it in a jar of water in which has been dissolved a quantity of magnesium sulphate sufficient to raise the density of the fluid to the point required to suspend the cerebral mass. Its specific gravity is then found by testing with the hydrometer, a difference of half a degree in the density of the fluid being indicated by the rise or fall of the substance immersed. (Bucknill.) The specific weight of the gray substance, whether cortical or ganglionic, is less than that of the white. The following is a table by Baistrocchi of the mean specific weights of the brain and spinal cord :

	Males.	Females.
White substance of the hemispheres	1.0273	1.0289
Gray substance of the hemispheres	1.0206	1.0239
Entire brain	1.0265	1.0338
Mantle or cortex	1.0278	1.0286
Striatum and thalamus	1.0453	1.0446
Midbrain and cerebellum	1.0387	1.0348

Hardening and Transportation of the Brain.—Most authorities are agreed that the best method of preparing the brain for subsequent gross and microscopical examination is to put it into two or three gallons of Müller's fluid * upon several layers of filtering paper; the fluid should be changed about every other day for a week, and after this at least once a week. In three or four weeks the brain will be consistent enough to be dissected without injury, and about this time it can be transported without much likelihood of harm. For the purpose of transportation a tin pail slightly larger than is absolutely necessary for the bulk of the brain can be used. The brain is enveloped in absorbent cotton; the pail is filled with Müller's fluid, and is then soldered and packed in a wooden box. (Meyer.)

Preservation of Brains as Gross Specimens.—Various methods of preserving the brain for gross study and as museum specimens have been recommended. If the brain is fresh, Giacomini's method is to place it at once in a saturated solution of zinc chloride. If the subject has been dead for some time, about sixty grammes of the solution are injected through the carotids, using slight pressure only. After twenty-four hours the leptomeninges are removed. The specimen can be kept in the zinc solution from four to six months, and is then transferred to methylated spirit, in which it is kept for two weeks, after which it is placed in pure glycerin, in which it may remain for from three to four months. On removal it is wrapped loosely in a cloth and allowed to dry, and when dry it is varnished. Blackburn hardens the brain in Müller's fluid for five weeks, then washes it in water and passes it through alcohols of increasing strength (for dehydration), finishing in absolute alcohol. The specimen is then transferred to a saturated solution of Japanese wax in chloroform, in which preparation it remains, occasionally changing, until the alcohol is thoroughly displaced by the chloroform. The preparation is next transferred to melted Japanese wax kept at a temperature near the boiling point, in which it remains until thoroughly infiltrated. A hemisphere after thorough dehydration may remain at least three days in each of the two solutions last men-

* Müller's fluid consists of potassium bichromate, 2½ parts; sodium sulphate, 1 part; distilled water, 100 parts by weight. It is best made a few days before it is used, and should be filtered.

tioned. When the infiltration is complete, the preparation is removed, the wax is drained off the surface, and it is allowed to cool. After cooling, the preparation is varnished. The surface may be painted or lettered, if desired. Fish, of Cornell University, recommends the following fluid: water, 400 c.c.; ninety-five per cent. alcohol, 400 c.c.; glycerin, 250 c.c.; zinc chloride, 20 grammes; sodium chloride, 20 grammes. The specific gravity of the mixture should be about 1.04, that is, a little greater than that of the brain itself. It is recommended that the cavities in the brain be filled with the mixture (cœlinjected), and, if practicable, the bloodvessels should also be injected. After an immersion of about three days, the specimen should be transferred to equal parts of the foregoing mixture and seventy per cent. alcohol for a week or more, in which liquid, on account of the lesser specific gravity, it should rest upon a bed of absorbent cotton. The specimen is stored in ninety per cent. alcohol. After dehydration in repeated changes of ninety-five per cent. alcohol, the brain is immersed in turpentine three parts and castor oil one part until it is translucent, changing the solution if it becomes cloudy. After this it is transferred to pure castor oil for a week or two, when it is allowed to drain until the surface dries on a layer of cotton covered with absorbent paper, and then it is painted a few times with an alcoholic solution of bleached shellac. The brain sections or dissections should be made before immersing in the turpentine oil mixture. It will be found that the alba becomes translucent first. The preparation at this particular stage may be put into the pure castor oil until thoroughly penetrated, and subsequently drained and shellacked.

Formalin Preparations.—In specimens in formalin, as in those for which osmic acid is used, the axis cylinders are well preserved. Formalin will even penetrate and fix the fibres of large nerves like the sciatic. Kitchell and Cullen have each given the results of their experience and some suggestions as to the preservation of specimens by formalin, which is a proprietary preparation composed of a forty per cent. solution of formaldehyde. Kitchell used one, two, ten, twenty-five, fifty, and one hundred per cent. of the commercial solution. His best results were obtained with the last three, the axis cylinders remaining entirely or almost entirely unshrunken. The specimens hardened in formalin he stained with eosin, fuchsin, and other aniline dyes, and also with Gage's hematoxylin, all of which colored the connective tissue strongly, while the unshrunken axis cylinders were but lightly stained. The neurokeratin network of the myelin was brought out much better than with Müller's fluid. Formalin, according to Cullen, is rapid in its action, and gives firm tissues without the contraction of the specimen often seen when alcohol is used. He found that he could use formalin in making excellent permanent specimens from frozen sections. The tissue is

first frozen with carbonic acid or ether, and then is cut, and placed in a five per cent. watery solution of formalin for from three to five minutes or longer, in fifty per cent. alcohol for three minutes, and in absolute alcohol for one minute. The tissue is now ready for mounting and staining in the usual way. Freezing destroys the blood in the section, and Prof. Welsh proposed to overcome this by first using the formalin and then freezing the specimen. The tissue is placed in a ten per cent. solution of formalin for two hours. The great value of a method like the freezing and formalin method just described is that it enables an opinion to be given on a specimen—a brain tumor, for instance—within fifteen minutes after it comes into the hands of the microscopist.

Golgi's and other Methods of Preparation and Staining.—One of Golgi's methods is to harden the entire brain or blocks of brain substance of about one to one and a half c.c. in size in a two per cent. potassium bichromate solution, the strength of which may be afterwards increased to three per cent. From six to thirty days are necessary to complete the hardening, the solution being frequently changed. The specimens are next placed in one half or one quarter to one per cent. solution of perchloride of mercury, in which they must be retained for eight days when the blocks are small, and at least two months for the entire brain. The solution should be renewed as often as it becomes yellow, and, according to Golgi, the pieces may be kept in it an indefinite time. The nerve fibres under the influence of mercury become quite colorless, while the cell bodies stain black. Subsequent washing of the sections in sodic sulphide solution renders them darker. The sections should be clarified and mounted in some balsamic medium, or they may be clarified in solution of ammonia. (Hamilton.) Another of Golgi's methods is to place small portions of the nervous system of embryos in a mixture containing potassium bichromate four parts and perosmic acid one part. After remaining in this solution for several days, the blocks of tissue are washed in a one quarter per cent. solution of silver nitrate and finally placed for two days in a three quarter per cent. solution of this salt. They are then ready for embedding either in paraffin or in celloidin. The method has its disadvantages, among which is the fact that all the nerve structures are not equally and simultaneously stained. (Turner.) Andriezen, Berkley, and others have modified the methods of Golgi in their studies of the human brain. Other methods are sublimate and alcohol fixation and hardening followed by staining in various anilines, especially toluidine blue; the fresh method of freezing and staining with aniline blue-black, after Bevan Lewis; Weigert's method for myelin; stains with acid rubin, and Nissl's stain with methylene blue, descriptions of which and of other of the newer methods will be found in the publications of Lenhossék, Andriezen, Berkley, Goodall, and Piersol.

CHAPTER V.

DISEASES OF THE ENCEPHALIC VESSELS, AND THE VASCULAR DISTURBANCES AND DISEASES OF THE BRAIN.

THE ENCEPHALIC CIRCULATION.

SOME general facts with reference to the blood supply of the nervous system—encephalic, spinal, and peripheral—have been given in Chapter I., where it is shown that both in the brain and in the spinal cord are found surface and central systems of vessels; in the brain certain arteries being distributed to its ganglia and core of white substance, and others to its cortex and subcortex. The method of termination of these arteries is shown in Fig. 105 on page 96. The arterial supply of the dura has been briefly considered in Chapter III. It is necessary to present in greater detail the anatomy and physiology of the encephalic circulation before taking up the diseases of the brain, most of which are of vascular origin. Descriptions of the cerebral circulation, cortical and central, are almost invariably drawn from the works of Duret, whose first communications on the subject were made in December, 1872, and in January, 1873, and were followed by a systematic treatise on the brain circulation. Almost exactly contemporaneous with Duret's were the publications of Heubner. These observers arrived at nearly the same results, and a dispute as to priority arose. Anatomists previous to the time of Duret and Heubner were content to indicate simply the lobar distribution of the encephalic arteries and veins; but these investigators traced and described the blood supply of the principal convolutions. Few real additions have been made since their publications, although special articles of considerable value have been published. One of these was by Browning, in 1889.*

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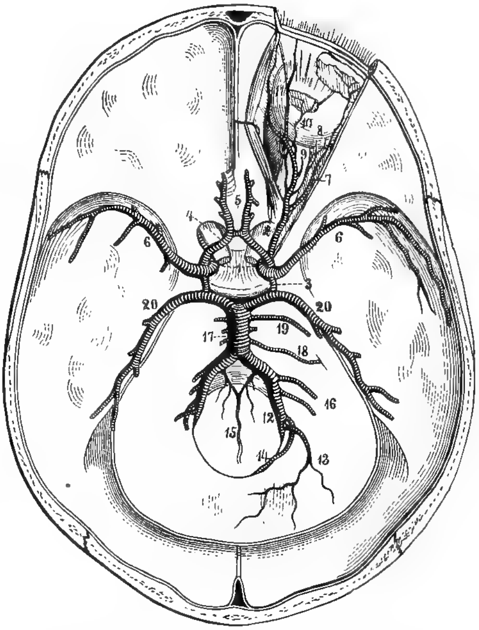
* Browning adopts the nomenclature of Wilder for his descriptive terms. The following from a table by him gives, for the vessels to which it will be necessary to refer, the common Latin names and the proposed English and Latin names:

Common Latin Names.	Proposed Names.	English Paronyms.
Cerebralis anterior.	Præcerebralis.	Precerebral.
Cerebralis media.	Medicerebralis.	Medicerebral.
Cerebralis posterior.	Postcerebralis.	Postcerebral.
Communicans anterior.	Præcommunicans.	Precommunicant.
Communicans posterior.	Postcommunicans.	Postcommunicant.

General Arrangement and Connections of the Encephalic Arterial Systems.—The general arrangement of the main trunks of the arteries of the interior of the cranium is shown in Fig. 262. Although the vessels at the base of the brain freely communicate, the branches given off to the two hemicerebrums are so completely separated that under usual conditions anything in the blood current of one side will not be carried into the circulation of the other. It has been found that when one carotid is tied the pressure in the ophthalmic artery on this side sinks very considerably, while that on the other side remains unaltered, a fact important to remember in connection with ligature of the carotid. The classical arrangement of the well known circle or polygon of Willis is, according to Browning, one precommunicant, two precerebrals, two medicerebrals (or two carotids), two postcommunicants, and two postcerebrals (Fig. 263). Important deviations from this arrangement often occur. Windle found that it holds in only a little more than half the cases recorded. Many isolated instances of interesting anomalies have been reported, as one by Lloyd, in which the basilar artery was a continuation of the left vertebral (Fig. 264). The basilar sent off on the left side as usual the postcerebral artery, but the right postcommunicant was one large continuous vessel from the foramen magnum to the carotid. The left internal carotid then divided into the medicerebral and the precerebral, and the precerebral of both sides sprang from this one trunk. A small vessel ran between the right medicerebral and one of the precerebrals. Practically more than one half of the brain was supplied from one side.

Fig. 262.



Arteries of the interior of the cranium: 1, internal carotid; 2, ophthalmic; 3, postcommunicant (posterior communicating); 4, precerebral (anterior cerebral); 5, precommunicant (anterior communicating); 6, medicerebral (middle cerebral); 7, lachrymal; 8, short ciliary; 9, central retinal; 10, muscular; 11, frontal and nasal; 12, vertebral; 13, postdural (posterior meningeal); 14, posterior spinal; 15, anterior spinal; 16, postcerebellar (inferior cerebellar); 17, basilar; 18, internal auditory; 19, precerebellar (superior cerebellar); 20, postcerebral (posterior cerebral.) (Bourgery.)

Cortical Distribution of the Main Cerebral Arteries.—The Sylvian or medicerebral artery physiologically and pathologically is by far the most important of the cerebral arteries. Its course is by way of the Sylvian fissure, dividing as it passes over the insula into four important branches, which are differently named and enu-

FIG. 263.

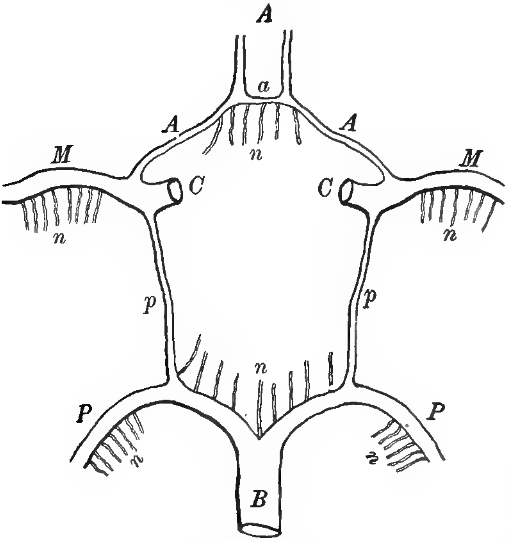
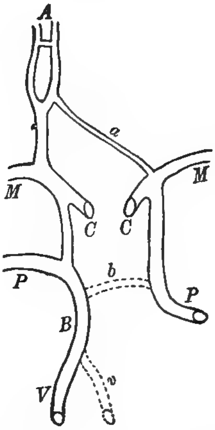


Diagram of the circle of Willis: *A*, *A*, *A*, precerebral arteries; *B*, basilar artery; *C*, *C*, carotid arteries; *M*, *M*, medicerebral arteries; *P*, *P*, postcerebral arteries; *a*, precommunicant artery; *p*, *p*, postcommunicant arteries; *n*, *n*, *n*, *n*, *n*, *n*, nutrient arteries.

FIG. 264.



Anomaly of the circle of Willis: *A*, precerebral artery; *B*, basilar artery; *C*, *C*, carotid arteries; *M*, *M*, medicerebral arteries; *P*, *P*, postcerebral arteries; *V*, left vertebral artery; *a*, small vessel replacing the right medicerebral artery; *b*, absent right branch of the basilar artery; *v*, absent right vertebral artery. (Lloyd.)

merated. The course of each of these branches and subdivisions is shown in Fig. 265, the nomenclature followed being that of Wilder and Browning. The precerebral or anterior cerebral arteries at their origins are almost perpendicular to the carotids, and are therefore horizontal in their courses. One precerebral is often larger than the other. They pass dorsad of the optic nerves into the great longitudinal fissure, where they are side by side. They pass along the longitudinal fissure on the mesal surface of the hemisphere until they turn backward around the callosal knee and continue on the dorsal surface of the callosum, sending cortical branches to mesal areas of the prefrontal, frontal, and parietal lobes. A terminal branch, the callosal, is also given off through the callosum and may be traced as far as the splenium. The postcerebral or posterior cerebral artery is one of the branches of the basilar, which bifurcates into the two postcerebrals just beyond the origin of the supercerebellar arteries. The postcerebrals turn outward around the midbrain, then pass backward, and eventually reach the ventral surface

of the cerebrum. Besides its central and basal branches, each post-cerebral has three main cortical branches, the temporal, the calcarine, and the parieto-occipital (Cunningham), called by Browning the pretemporal, the post-temporal, and the occipital. The temporal branches go to the under surface of the temporal lobe; the calcarine follows the calcarine fissure; and the parieto-occipital

FIG. 265.

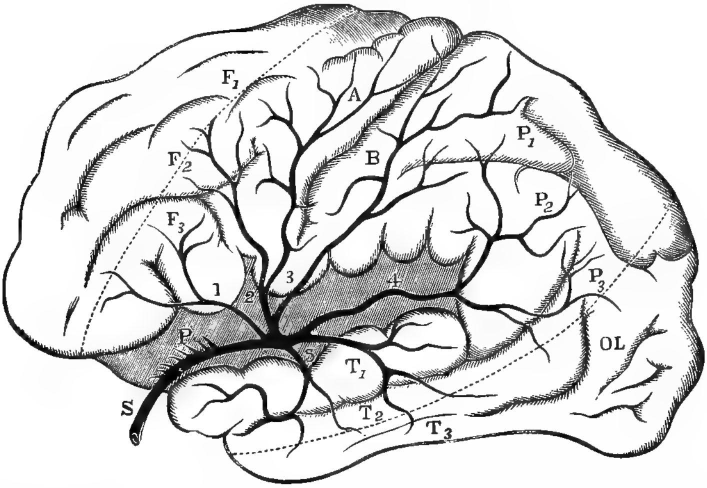


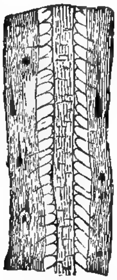
Diagram showing the distribution of the medicerebral artery: S, Sylvian or medicerebral; P, perforating branches; 1, subfrontal branch, 2, precentral, 3, postcentral, 4 and 5, parietotemporal and temporal branches; A, precentral convolution; B, postcentral convolution; F_1, F_2, F_3, superfrontal, medifrontal, and subfrontal convolutions; P_1, P_2, P_3, parietal, subparietal, and angular convolutions; T_1, T_2, T_3, supertemporal, meditemporal, and subtemporal convolutions; OL, occipital lobe. (After Duret.)

follows the parieto-occipital fissure. The cuneus is chiefly supplied by the calcarine, which also supplies the lingual convolution; the parieto-occipital sends branches to both the cuneus and the precuneus. The calcarine vessel is comparatively large, and is sometimes the seat of serious arterial hemorrhage.

Subdivisions of the Cortical Arteries.—The cortical arteries subdivide into secondary and tertiary branches, and from the latter and also from the main trunks is formed a system of arborizations. From this network of vessels, which is in the plane of the pia, nutrient arteries penetrate the cerebrum perpendicularly. A section of one of the arteries is seen in Fig. 266. These vessels are of two kinds, long and short. The former penetrate the alba or centrum ovale until they approach the central arterial system, with which, however, they do not communicate. This gives on the confines of the cortical and central domains a kind of neutral zone, where nutrition proceeds less actively. It is the frequent seat of the lacunar softenings in the aged. The short cortical arteries, which are also

more slender than the others, terminate in the cortex or just at its junction with the alba, their capillary branches and those from the long arteries forming a network. This is arranged in layers. The first or outer layer contains but few vessels; the second has a compact vascular network made up of extremely fine polygonal meshes; internally the meshes become larger, and in the medullary substance the meshes are still wider and lengthened out vertically. The cortex and subjacent white matter are liable to be affected together, as their vessels in both cases are derived from the arteries of the pia. (Charcot and Duret.)

Fig. 266.



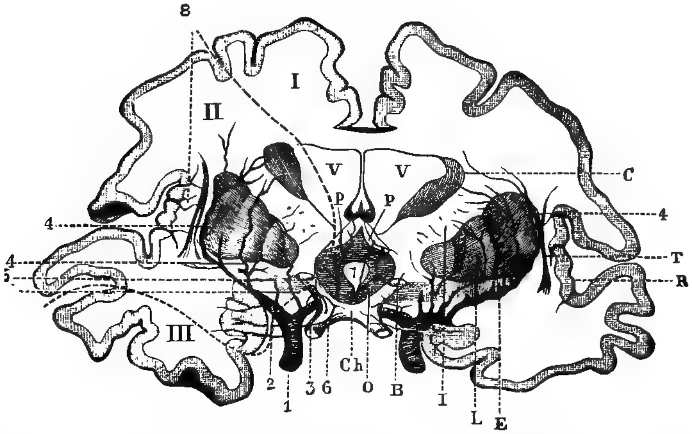
An artery from the cortex cerebri in longitudinal section; numbers of fine fibres are seen streaming into the brain substance. (Obersteiner.)

Intercommunication of Cortical Arterial Territories.—Branches of special cortical vessels freely anastomose, but between the different cortical systems, anterior, middle, and posterior, communication is not free. Duret held that the different systems were almost absolutely distinct. Cadiat opposes various objections to the view of Duret, having found that even in making a gentle injection into any branch of the circle of Willis, blood is distributed to the cortex of the entire hemisphere. Duret contended that the diameters of the anastomosing vessels do not exceed one fourth of a millimetre, while Cadiat and Heubner allow them a calibre of a full millimetre. A. J. Parker and the author found in two brains carefully injected that communications between the three vascular territories were freer even than is held by Heubner, some of the communicating vessels being at least two millimetres in diameter. Great individual differences may exist.

Distribution of the Central Cerebral Arteries.—The arteries which go to the basal ganglia, capsules, and centrum ovale are derived from the circle or polygon of Willis. They dip into the brain substance in the anterior and posterior perforated spaces (precribrum and postcribrum). The head of the caudate nucleus is supplied by anteromedian branches which leave the precerebral artery shortly after it branches from the internal carotid. The most important set of interior vessels is that which leaves the medicerebral artery close to its origin, and, passing into the brain substance, supplies all of the caudatum except the cephalic extremity, the entire lenticular body, the internal capsule, and a large portion of the thalamus. From the postcerebral arteries or from the basilar artery near its bifurcation a posteromedian group of vessels is given off to irrigate the inner aspect of the thalamus. The posterolateral branches originating in the postcerebral are distributed to the external and posterior regions of the thalamus, the choroid plexus, the wall of the lateral ventricle, the quadrigeminal body, and the dorsal portions of

the crura. These distributions are shown in part in Fig. 267, particularly the branchings of the middle cerebral artery. The lenticulostriate and lenticular arteries are of the most importance in the study of intracerebral hemorrhage. The lenticulostriate artery is most frequently the seat of intracerebral hemorrhage, and has been called by Charcot the artery of cerebral hemorrhage. The branch which supplies the third segment of the lenticula, the superior por-

FIG. 267.



Transection of the cerebral hemispheres about one centimetre behind the optic commissure: Ch, chiasm; B, section of the optic tract; L, lenticula; I, internal capsule; C, caudatum; E, external capsule; T, claustrum; R, insula; V, V, section of the lateral ventricle; P, P, fornicolumns; O, cinerea of diacœle (third ventricle); I, precerebral, II, medicerebral, III, postcerebral arterial territories; 1, internal carotid, 2, medicerebral, 3, precerebral arteries; 4, 4, lenticulostriate arteries; 5, 5, lenticular arteries. The optostriate artery is not represented in the figure. (After Duret.)

tion of the internal capsule, and the caudate body is particularly likely to be ruptured. The cerebellum is irrigated by arteries from the vertebral and basilar arteries. The postcerebellar or inferior cerebellar arteries are usually derived from the vertebrals; the basilar gives off the medicerebellar and anterior cerebellar branches; and cephalad the basilar subdivides into the two postcerebral arteries.

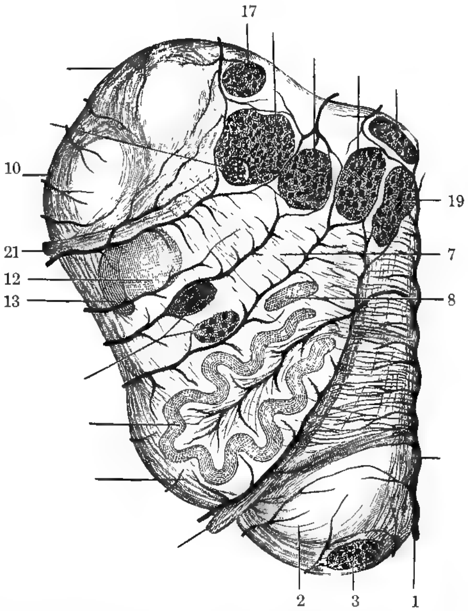
The Large Arteries of the Pons and Oblongatas.—Lesions of the pons and oblongatas, as of other parts of the encephalon, are largely diseases of the circulation or of the walls of the vessels,—hemorrhages, embolisms or thromboses, aneurisms sufficient in size to produce definite symptoms, other arterial enlargements, as periarterial nodes, and tubercular deposits along the vessels. The vertebral arteries, and the basilar artery, which is constituted by their fusion, are the direct or indirect sources of all the blood to the pons and oblongatas. The vertebrals may vary somewhat in volume, and special variations have been described by many anatomists. The left is commonly more voluminous than the artery on the right side.

The vertebral on this side usually arises about three quarters of an inch (two centimetres) from the innominate, but sometimes its origin may be closer to their bifurcation. As the left vertebral arises from the subclavian when the latter is still vertical or nearly so, while the right springs from the horizontal portion of the subclavian, emboli are more likely to be carried by the circulation to the left side of the bulb, just as a left carotid because of its more direct origin from the aorta is more likely to carry vegetations to the arterial system of the left half of the cerebrum. Numerous variations in the places of origin and in the branches of the vertebral arteries have been described by anatomists, and are of most importance in connection with surgical operations. The vertebral artery passes through the foramens in succeeding costotransverse processes of the cervical vertebræ from the sixth upward as far as the axis, where it first turns outward and then again upward through the foramen to the transverse process of the atlas, after which it turns backward behind the articular process, lying in the groove of the posterior arch of the atlas. Piercing the occipito-atloid ligament and the dura, it enters the cranium through the foramen magnum. At first the two vertebrals are placed along the sides of the postoblongata, but as they pass cephalad they converge, and just at the border of the pons unite to form the basilar. The basilar generally corresponds in length to the length of the pons. At its cephalic extremity it gives off nearly at right angles the two postcerebral arteries. Not infrequently the basilar makes a slight bend towards the right. Near the middle of the pons it gives rise to two medicerebellar arteries. The postcommunicant arteries which spring from the postcerebrals, as already described, unite the vertebral and carotid systems. The anterior spinal artery is formed by the fusion of two intracranial branches of the vertebrals, which arise near the termination of the latter about the junction of the postoblongata and pons, and descend on the ventral surface of the postoblongata to unite at the level of the foramen magnum. The single artery thus formed passes caudad along the ventral surface of the spinal cord. The posterior spinal arteries arise from the vertebrals at the sides of the postoblongata, and, passing caudad, turn towards the dorsal surface of the spinal cord, along each side of which they descend as a pair. It is therefore a point in applied anatomy worthy to be remembered that lesions such as emboli and thrombi of oblongatal branches of the vertebrals may give rise to spinal as well as bulbar symptoms.

Arterial Supply of Special Regions of the Bulb.—Small branches are given off to the bulb from the intracranial portions of the anterior and posterior spinal arteries; also directly from the vertebral and the basilar and to a less extent from the postcerebral and postcerebellar arteries. Some enter the pons and oblongata by

the ventral median fissure; others by the sulci or furrows between the pyramids, restiform bodies, and other structures of this region. Duret divides the arteries of the pons and oblongatas into *lateral*, chiefly destined to the nerve roots; *median*, which go to the nuclei of the floor of the fourth ventricle; and *special* vessels, which go to the other constituent parts of the bulb, as to the olives, pyramids, fillet, and trapezium. In the median plane the arteries to the nuclei, which are arranged in several groups of increasing extent from below upward, form a sort of scaffolding whose divisions are parallel. They are nearly capillary, and show no naked eye anastomoses. The oblongatal distribution of each anterior spinal artery is to the nuclei of the spinal accessory, hypoglossal, and inferior facial nerves; the lower and median protuberantial or pontile arteries are destined for the pneumogastric, glossopharyngeal, and hypoglossal nuclei; while the upper protuberantial arteries send their branches to the nuclei of the third and fourth nerves. The postcerebellar arteries supply chiefly the lateral and dorsal portions of the pons and preoblongata. According to Adamkiewicz, the exact distribution of some of these arterial branches is by way of the arteries of the lateral sulci or furrows to the pyramids and their decussation, to the fillet, to the raphe, interolivary layer, nucleus arciformis, inner accessory olive, olivary body, and especially to the hypoglossal nucleus; and by the arteries of the median fissure to the posterior columns, nucleus gracilis, and especially to the nucleus of the accessorius. Special branches supply the nucleus gracilis and cuneatus, substantia gelatinosa, nuclei of the lateral columns, superior and inferior olives, and the acoustic

Fig. 268.



Horizontal section of the oblongata, showing the distribution of the arteries: 1, artery entering the median fissure and raphe; 2, pyramid; 3, arciform nucleus; 4, olive; 5, olivary nucleus; 6, arciform fibres; 7, reticular formation; 8, accessory olivary nucleus; 9, lateral nuclei; 10, 11, arciform fibres of the restiform body; 12, substantia gelatinosa; 13, descending root of the trifacial nerve; 14, dorsal longitudinal bundle; 15, facial nucleus in the terete funicle; 16, external facial nucleus; 17, inferior portion of the auditory nucleus; 18, vagus nucleus; 19, 20, hypoglossal nuclei; 21, vagus nerve; 22, hypoglossal nerve. The black branching lines represent the chief arteries. (Leidy.)

nucleus—these arteries running through the upper part of the restiform body. The nuclei of the glossopharyngeus, vagus, and hypoglossus also receive special branches.

Veins of the Brain.—Brief reference will next be made to special venous territories, cerebral, cerebellar, and bulbar. A venous circle or polygon, corresponding closely to the circle of Willis, is formed at the base of the brain, the two basilar veins being connected with each other by a postcommunicant vein, while the precerebrals are united by a precommunicant vein. The veins,* like the arteries, can be subdivided into cortical or superficial, and ganglionic or central. The former gather blood from the capillaries of the cortex and carry it to the sinuses. These veins are usually divided into three groups: the veins of the internal surface of each hemisphere, the *internal cerebral veins;* the veins of the external face of the hemispheres, the *external cerebral veins;* and the veins of the inferior face of the hemispheres, the *inferior cerebral veins.* The internal cerebral veins empty

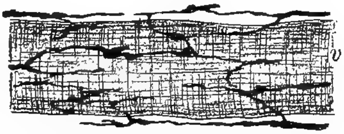
* The following table by Browning enumerates the most important of the encephalic veins, with their common Latin names and the names suggested by Browning and Wilder:

Common Latin Names.	Proposed Names.	English Paronyms.
Cerebri superior.	Supercerebralis.	Supercerebral.
Cerebri anterior. Callosi anterior externa.	Precerebralis.	Precerebral.
Cerebri media.	Medicerebralis.	Medicerebral.
Fossæ Sylvii.	Sylvii.	Sylvian.
Basilaris Rosenthali. Cerebri inferior s. ascendans.	Subcerebralis.	Subcerebral.
Occipitalis lateralis. Cerebralis lateralis et inferioris.	Postcerebralis.	Postcerebral.
Occipitalis interna. Cerebri posterior inferior.	Suboccipitalis.	Suboccipital.
Callosi posterior externa.	Callosalis.	Callosal.
Magna Galeni. Cerebri interna communis.	Galeni.	Galen's.
Cerebri interna s. intima s. profunda.	Velaris.	Velar.
Corporis striati. Lateralis ventriculi.	Tænialis.	Tenial.
Cornu anterioris.	Precornualis.	Precornual.
Septi lucidi. Anterior ventriculi.	Paraseptalis.	Paraseptal.
Cornu posterioris.	Postcornualis.	Postcornual.
Choroidea.	Medicornualis.	Medicornual.
Medullares superiores.	Superalbales.	Superalbal.
Rami corporis striati.	Striatales.	Striatal.
Rami thalami optici.	Thalamici.	Thalamic.
Ramus thalami optici profundus (Rosenthal). V. pedunculi cerebri.	Thalamocruralis.	Thalamocrural.
Azygos conarii.	Conarialis.	Conarial.
Cerebelli superior media.	Supercerebellaris.	Supercerebellar.
Cerebelli superior lateralis.	Paracerebellaris.	Paracerebellar.
Cerebelli inferior.	Subcerebellaris.	Subcerebellar.

for the most part into the longitudinal sinus. A few of them go to the falcial or inferior longitudinal sinus, into the vein of Galen, or into the precerebral vein. The external cerebral veins are partly ascending and partly descending, the former emptying into the longitudinal sinus and the latter into the lateral, superpetrous, and cavernous sinuses. Among the veins of the external surface one more voluminous than the others is situated at the level of the central fissure, opening partly into the longitudinal sinus and below partly into the cavernous or the superpetrous sinus. This is the *large anastomotic vein of Trolard.* The occipitotemporal veins of the inferior surface of the cerebrum empty into the lateral sinus, and those of the inferior portions of the frontal lobe into the precerebral veins, the middle cerebral vein of the insula, and the beginning of the longitudinal sinus. Certain groups of central or ganglionic veins are tributary to the veins of Galen. These are the basilar veins, the veins of the striatum, thalamus, callosum, conarium, choroid plexus of the third ventricle, postcornua, and postgeminum. After the two veins of Galen unite into the central great vein, this trunk receives veins from the dorsal surface of the cerebellum, and from one of the suboccipital veins of each side. The veins of the striatum, in addition to their own tributaries, receive small veins from the septum, fornix, and precornu. The basilar veins are formed by the confluence of the deep Sylvian, inferior striate, and some small inferior cerebral veins. Running backward, they enter the veins of Galen on each side just before their union into one trunk.

Nerves to the Bloodvessels.—Nerves are distributed to both arteries and veins, although little is known of the neural supply of the latter. Nerves to the capillaries have not yet been discovered. Much has been written about the nerve supply to bloodvessels, but demonstrated facts are comparatively few. Usually these nerves are divided into *vasoconstrictors* and *vasodilators.* A monarchical vasomotor centre is situated somewhere in the oblongata, and subsidiary centres are placed at various levels of the spinal cord. Ott and others have located additional vasomotor centres in the thalamus and cortex. Some of the intracranial vasomotor nerves, both vasoconstrictor and vasodilator, probably proceed directly from the oblongatal centres; others from the thoracic region of the cord. Thalamic and cortical centres exert an influence not yet thoroughly understood over the centres in the oblongata and cord. Lesions of the cortex, of the thalamus (probably also of the striatum), of the pons and oblongata, and of the spinal cord, especially of its thoracic part, may give rise to definite vasomotor diseases—affections which manifest themselves

Fig. 269.



Nerve fibres accompanying a small artery (*v*) from the mesentery of a rabbit; gold preparation (Piersol.)

by phenomena due on the one hand to constriction or spasm, or, on the other, to relaxation or paresis of the vessels. The vasomotor nerves terminate in the muscular coat of the arteries. Some physiologists believe in only one set of them, holding that they give rise to phenomena of constriction or dilatation according to the manner in which they are affected.

Character of the Cerebrospinal Fluid in Different Diseases.—The chemical composition of the cerebrospinal fluid obtained by puncture after Quincke's method may give information as to the character of the cerebral or spinal disease. In tumor the fluid is less albuminous than in tubercular meningitis, although an exact decision cannot be reached. In six cases of tumor the proportion of albumen varied from a trace to 0.8 part per 1000. In six cases of tubercular meningitis it varied from 0.5 per 1000 to 1.6 parts per 1000. Quincke claims to have found seven per cent. of albumen in a case of tumor. In a case of abscess 0.7 part per 1000 was found. The inflammatory fluid always coagulated in part, while that from the tumor cases rarely did so. In fluid from tumor cases sugar was invariably found by delicate tests; in inflammatory fluid it was rarely found. Puncture may also give evidence as to the physical characters and bacterial contents of the fluid. In three cases streptococci and in one the pneumococcus was found; the tubercle bacillus was detected in the evacuated cerebrospinal fluid in four cases out of six of tubercular meningitis examined.

BRAIN PRESSURE.

The term *brain pressure* is usually employed to describe the compression to which the brain is subjected, under normal or abnormal conditions, by the cerebrospinal fluid. Variations of this pressure may cause special phenomena within the skull. The communication within the rachidian and cephalic cavities containing fluid is very free. Pressure applied to the subarachnoid space through an opening in the frontal portion of the skull is transmitted so that it can be readily recognized through an opening into any portion of the subarachnoid space. In spinal meningocele the subarachnoid pressure has been found to vary considerably in sleep, under narcosis, or during excitement, as when a child is crying. The methods employed by physiologists, such as the injection into the subarachnoid space of a three fourths per cent. salt solution, to experimentally increase brain pressure, produce the same results as those brought about by diseases such as meningitis, neoplasms, hemorrhages, or edema, which give rise to exudate and increase the volume of material within the skull. Among the phenomena of brain pressure, when consciousness is not lost, is pain in the head which is present in the early stages of compression but disappears when it becomes more complete. Naunyn and Schreiber attribute it to stretching of the dura and to anemia of the brain, the dura, as already shown, being supplied with sensory nerves.

This explains some of the forms of headache. Other important manifestations of brain pressure are convulsions, interference with respiration, and slowness of the pulse, which almost invariably occur after the pressure has reached a certain degree. Schulten found that so long as the brain pressure was less than the minimum blood pressure the former had no appreciable influence on the latter. The pulse becomes slow as the result of elevation of the blood pressure when the brain pressure rises beyond the minimal blood pressure. Some of the changes produced by increased intracranial pressure can be observed with the ophthalmoscope. The retinal arteries become narrowed, and hence the appearance is that of anemia, less blood flowing through the contracted vessels. Schulten made experimental observations on the condition of the ophthalmic arteries when brain pressure was increased, by inserting the canula of a mercury manometer into the vitreous, using a special mode of illumination. In hemorrhagic apoplexy the phenomena of brain compression largely dominate the scene, and they are also important in traumatisms, and in some of the affections accompanied by changes in the quality of the blood, as in hydremia. Respiration becomes irregular even before changes in the pulse are observed; the breathing, as the brain pressure rises, becomes labored and stertorous, and perhaps of the Cheyne-Stokes variety. The pulse becomes slow and full, although before a fatal issue it may again become rapid. Pupillary disturbances are common. Both pupils usually dilate. When the pupils of the two sides differ it is because of some local unilateral influence exerted by a lesion. Choked disks are often present. Some if not most of the clinical phenomena of brain pressure are due to accompanying anemia of the compressed brain substance. In treating a case of hemorrhagic apoplexy, brain tumor, or meningitis, with acute exacerbations of pain or other phenomena of increased intracranial tension, the conditions as to brain pressure must be always borne in mind. The views of almost equally good authorities as to the means of reducing brain pressure are diametrically opposed. Some, following the old school of venesectionists, advise free bloodletting to reduce arterial tension, while with better reason others bend their efforts to increasing blood pressure so as to relieve the cerebral anemia.

CONCUSSION, COMPRESSION, AND SPECIAL TRAUMATISMS OF THE BRAIN.

Concussion of the Brain.—Concussion of the brain and compression of the brain belong properly to works on surgery, but it may serve a good purpose to direct attention briefly to these affections because of the relations they bear to such intracerebral diseases as hemorrhage, tumor, and abscess, and because questions concerning them frequently arise in connection with cases of the so-called traumatic neuroses, traumatic hysteria, the railway spine, and similar

affections claiming the attention of the medical jurist. Concussion of the brain has been divided into (1) encephalic vibration without visible lesion; (2) vibration with extravasation of blood; (3) vibration followed by serosanguinolent transudation; and (4) vibration with laceration of the brain substance. (Agnew.) The last three of these classes include cases recognized by all as of frequent occurrence. Regarding the first class, it has often been questioned whether, strictly speaking, true concussion of the brain is ever without recognizable lesion. Some still believe with Agnew, Erichsen, and others in the doctrine of pure molecular vibration as applied to both brain and cord. According to this view, the molecules of nerve substance are jarred and functionally injured in much the same way as a magnet is partially demagnetized by a sudden and severe blow. As more is learned, however, about traumatic lesions, minute and massive, fewer cases need to be relegated to simple vibration. A jar sufficient to vibrate the encephalic or spinal masses will in the vast majority of cases cause at least minute vascular lesions. Among the direct effects of concussion of the brain may be disturbances of cell processes and cell bodies, which will before long possibly be recognized by the microscope.

Compressibility of the Brain Substance.—It was long held that the brain substance was incompressible, and that apparent diminutions in the space occupied by it were due to the displacement of the cerebrospinal fluid. Adamkiewicz, among others, denies this assumption. He produced compression in the brains of animals by laminaria, at the same time measuring the arterial pressure. He considered that the brain substance can be compressed, especially by slowly developing pathological areas. The lessening volume of the brain being attained by the cerebral fluids leaving the cranial cavity through the blood and lymph vessels, there is less condensation of the nerve tissue proper. The "intracranial pathological area" grows at the expense of the brain substance, and not at the expense of the cerebrospinal fluid, which is not displaced. In regard to anemia of the brain, he proved by examination of compressed brains that the development of an "intracranial area" has no influence upon the lumina of cerebral capillaries. He found that they not only did not diminish in size, but dilated. Long continued pressure of the brain with laminaria resulted in the development of small bloodvessels even in the parts compressed. The blood circulation of the brain is increased under these circumstances. The following phenomena of compression were observed: attacks of contralateral spasms with disturbances of consciousness; trophic disturbances of the eye, and disorders of innervation of the ocular muscles; spasms with increased tendon reflexes, and tremor of half of the body opposite the compressed hemisphere; and finally paraplegia.

Diagnosis of Concussion from Compression.—Usually it has

been held that cases of concussion and compression can be separated; but this is certainly doubtful for many cases. A critical study of the tables of differential diagnosis between concussion and compression simply shows that in compression there is greater interference with or alienation of the bodily functions,—a greater impairment of consciousness, of the special senses, of motility, respiration, pulse, temperature, and of the functions of the abdominal and pelvic viscera.

Experiments and Conclusions of Duret.—Duret showed that the skulls of animals are elastic or pliable, and when they strike upon resisting objects or when they receive a violent blow, if fractures do not result, or perhaps in some cases with fractures without displacement, the skull is momentarily depressed, and thus the capacity of the cranial chamber is transiently diminished. The cerebrospinal liquid is driven in various directions, waves of movement being communicated to the fluid both outside and within the ventricles. The fissure of Magendie is the chief channel of communication between the intraventricular and extraventricular chambers. One effect is to drive the cerebrospinal fluid through the iter into the fourth ventricle. Small hemorrhages result from the distention produced by the liquid, and are especially to be seen in the vicinity of the iter and in the metepicœlian floor. Such hemorrhages are, however, common everywhere in the membranes and in the substance of the brain. Laceration sometimes occurs opposite the point of contact, and in this vicinity hemorrhages may be abundant. Vascular spasm with resulting anemia occurs over large areas of the brain.

Conclusions of Phelps.—Phelps has exhaustively studied in man the subject of traumatic intracranial lesions, the chief of which are hemorrhages, subarachnoid serous transudation, arachnitis, contusions, laceration usually with contusion, and pyogenic parenchymatous inflammation. The chief symptoms of hemorrhage from traumatism are an abnormal temperature, a complete or partial loss of consciousness, changes in the character or frequency of the respiration and pulse, disturbance or abrogation of muscular function, and irregularity of the pupils. These symptoms may of course be commingled with those of other conditions, such as contusion, laceration, and inflammation. Continuing abnormal temperature points to large and comparatively uncomplicated hemorrhage. Epidural hemorrhage is usually of this kind. The primary unconsciousness which is of frequent occurrence in cases of hemorrhage is a symptom of complicating general contusion; the secondary unconsciousness, due to the loss as well as the pressure of blood effused, follows with or without an interval of restored consciousness, dependent upon the severity of the diffused injury of the parenchyma and the rapidity of the hemorrhagic effusion. Consciousness is always lost in fatal cases; it is retained in fifty per cent. or more in recovering cases. The pulse may be normal, slow, or frequent in large ex-

travasations wherever situated; but frequency is much more usual in hemorrhage than in other intracranial lesions, so that it may be considered fairly diagnostic of hemorrhage, this being probably epidural. The bilateral variation in the force and fulness of the arterial pulsations is common to hemorrhages and to injuries of the brain substance, and of importance, therefore, only in general diagnosis. An alteration in the character or frequency of respiration is almost invariable in fatal cases in which hemorrhage is an approximately isolated lesion. General or local paralysis and disordered muscular action may be direct symptoms of hemorrhage compressing and irritating recognized centres of muscular control; tetanic spasm is not infrequent, but clonic contraction is of rare occurrence, except as the result of an associated lesion. Normal pupils or every possible condition of pupillary contraction or dilatation may be present. Contraction of one pupil, however, never occurs without some change in its fellow. Sensory disturbances, as delirium or irritability, are not symptoms of hemorrhage, and when they occur are to be regarded as indicative of an accompanying lesion of the parenchyma. Under dural hemorrhage and under meningitis of various forms, the symptoms of some of these traumatic lesions enumerated by Phelps have been considered. General contusion is a constant complication of all other forms of intracranial injury, but rarely occurs as an isolated lesion of fatal severity. Its symptoms are irregular and indefinite. A loss of consciousness at some time and in some degree is more nearly constant than any other individual symptom. Primary or early delirium, like primary unconsciousness, in both simple and complicated cases is to be ascribed solely to the influence of this lesion. Laceration is almost if not quite invariably complicated by a concomitant general contusion and by a resultant hemorrhage. Trivial cases may show no secondary symptoms which indicate laceration. The primary unconsciousness may be replaced by a condition of lethargy or blunted perception, passing through somnolence and coma into death. The primary stage is most frequently succeeded by mental aberration or decadence, which may terminate in recovery, in permanent dementia, or in death. In exceptional instances consciousness may remain unimpaired, with extensive laceration even of fatal import. The phenomena which directly point to laceration may be enumerated as certain peculiarities of temperature, psychic disturbance, loss of fecal and urinary control, and clonic convulsions. The pyogenic, parenchymatous inflammations and abscesses resulting from traumatism will be considered under encephalitis and abscess. In all forms of intracranial traumatisms, according to Phelps, in endeavoring to make a differential diagnosis the possible multiplicity of lesions must be kept prominently in mind, and the relative as well as the absolute value of symptoms must be estimated.

HYPEREMIA OF THE BRAIN AND ITS SOFT MEMBRANES.

Definition and General Considerations.—In hyperemia of the brain and of its soft membranes, which must be considered together, the encephalic vessels contain, for a time at least, an amount of blood disproportionate to that present in the rest of the system. Its existence as a clinical entity has been doubted; but certainly a hyperemia of passive character due to obstruction may occur, and it is almost equally certain that an active hyperemia may affect the brain as the result of anxiety, cerebral overwork, injuries, effects of the sun, and toxic substances circulating in the blood. It is true, however, that the diagnosis of hyperemia or congestion of the brain has too often been made. Recurring attacks of hyperemia form part of the clinical history of some forms of insanity.

Varieties.—Hyperemia may be either *acute* or *chronic*, *active* or *passive*. Other special varieties are sometimes described, as the light and the severe form, the cephalalgic, the psychic, the convulsive, and the apoplectic form. (Hirt.) Such varieties simply represent differences in the intensity of the pathological process. Localized and circumscribed hyperemia of the active type may occur in the vicinity of tumors, meningitis, or encephalitis.

Symptoms.—The symptoms of acute general hyperemia of the brain are headache, sensations of throbbing or confusion, tinnitus, phosphenes or photopsias, flushing of the scalp and face, and injection of the conjunctiva. The pupils are generally contracted. Insomnia and even delirium with hallucinations and illusions may be present in severe cases. In rare cases congestive apoplectiform attacks, in which consciousness is lost for a time, occur. The pulse is usually slow, hard, and full. The symptoms of static or passive hyperemia, dependent upon local obstruction or deficient cardiac and vasal energy, usually come on more slowly, are more continuous, are of less decided character, and are often associated with other evidences of disease, particularly of cardiac and pulmonary involvement. Dull headache, dizziness on exertion, and feelings of fulness in the head are common. Marked tinnitus, throbbings, and visual phenomena are not so frequently complained of as in the active forms of congestion. Attacks of spasm, with or without unconsciousness, are among the rare results of brain congestion of the apoplectiform type. The existence of chronic hyperemia is indicated by such symptoms as have just been described as belonging to the acute forms, but these are of less intensity and have marked exacerbations. As already stated, it is doubtful whether a persistent chronic hyperemia without organic change is present over a long period.

Etiology.—Active hyperemia may be the result of organic disease or of functional disturbances of the circulation from causes acting either within or outside of the body. Subacute and chronic

hyperemia of the brain, with acute exacerbations under exciting causes, sometimes accompanies hypertrophy of the heart, which in its turn may be dependent upon aortic valvular disease, or upon some extracardiac obstruction to the circulation. Sudden and widespread contraction of the arterioles in other parts of the body may cause temporary encephalic hyperemia, as when the vessels of the skin and other organs contract over large areas from exposure to extreme cold. A few drugs, as amyl nitrite and nitroglycerin, induce hyperemia of the brain by their action on the vasomotor centres. Some neurotics seem to have an inherent tendency to vasomotor weakness, leading to recurring cerebral hyperemia, generally of a mild type. The transient headaches of children may be accounted for in this way. Many physiological and clinical facts tend to prove that hyperemia of the surface of the brain may be the result of psychical overactivity. Tracings have been made with the sphygmograph by Mosso, Batty Tuke, Putnam-Jacobi, and other observers, where the brain has been exposed as the result of disease of the cranial walls, and these cases have shown bulging and increase of blood pressure due to mental activity or excitement. In other words, a functional hyperemia is thus produced, and is due either to direct action of the vasodilator centres or to retardation of inhibition of vasoconstrictor centres. Passive hyperemia of the brain is, as a rule, due to diseases which cause interference with or obstruction to the return of the blood within the capillaries of the brain, as aneurisms or tumors of the neck, weakness of the heart with fatty degeneration, and disease of the lungs. The playing of wind instruments is also a possible cause.

Pathology.—The intracranial cavity is closed and practically always filled, but, as shown when considering concussion of the brain, the usual state of balance between blood pressure and brain pressure—that is, the pressure of the cerebrospinal liquid—is at times altered. The fact that brain pressure may be changed makes it possible to have a cerebral hyperemia. According to Roy and Sherrington, the encephalic circulation is regulated by three factors, —the general arterial pressure, the general venous pressure, and the presence or absence of chemical products of cerebral metabolism contained in the lymph which bathes the walls of the arteries of the brain. These last may sometimes act locally, bringing about different degrees of cerebral activity. One of the most important facts observed by them was that the blood supply of the brain varies with the pressure in the systemic arteries. In chronic nephritis with increased blood pressure, cerebral congestion and hemorrhage are well known occurrences. One explanation of cerebral hyperemia is that it is dependent upon paralysis of the vasomotor nerves, which allows dilatation and overfilling of the bloodvessels. Such a condition of paresis or paralysis might be brought about by the circulation of

toxic agents in the blood, affecting the vasomotor centres either in the bulb or in the cortex. The ingestion of alcohol or other narcotics and products of metabolism in the body may give rise to hyperemia. The condition present in many cases of so-called hyperemia is certainly one of toxemia: the blood retained in the distended capillaries is changed in quality as well as in quantity. In passive hyperemia, for instance, the capillaries contain an excessive amount of carbon dioxide, the influence of which on the encephalic centres is more baneful than the mere presence of an undue amount of blood. The blood is not properly oxygenated; and in other cases special toxic agents may be dammed up in the congested capillaries. The congestion of the brain and its membranes which results from exposure to high temperatures is due to a toxic agent in the blood, the toxin being the product of catabolic changes produced by high temperature. (Tuke and Woodhead.) It is well known that uric acid increases arterial tension, and in this way conditions conjointly hyperemic and toxemic may be produced in the head, and in various parts of the nervous system. De Sarlo and Bernardini have recorded experiments on the cerebral circulation during psychical activity and under the action of certain intellectual poisons. The experiments were made upon a patient of a moderate degree of intelligence who had a fracture of the skull that permitted instrumental registration of the movements of the brain. The object of the investigation was to determine the relations existing between the cerebral pulse and the emotions. They found that in all emotional conditions there was an increase of the cerebral volume and of the height of the pulsations. The physical pain produced by the electric current always caused vasal spasm, while the pain induced by other means did not specially modify the circulation, or the modifications occurred at the same time with the other emotions. They conclude that the manner of reaction by the cerebral circulation produced by emotions is not altered by the effects of these agents, and that the changes in the cerebral circulation induced by them are of the nature of reflexes, not subject to modification by preexisting conditions. They offer the suggestion that as the psychic phenomena observed by them in their subjects were due to chemical intoxication, and not to circulatory disturbances, many forms of mental disease thus have for their cause, instead of cerebral hyperemia or anemia, some intoxication, of the real nature of which we are still in ignorance. (Soury.)

Pathological Anatomy.—Hyperemia of the brain is a vital process, and one which does not always leave appearances which can be detected after death, and the records of autopsies have in this respect been often misleading. Engorged veins and capillaries often seen at autopsies are sometimes set down as evidences of congestion. The brain passively congested during life may present an anemic appearance after death. Among the best evidences of cerebral hyper-

emia is the presence of minute extravasations in the floor of the ventricle and scattered throughout the cortex and the centrum ovale. Microscopical examination may show enlarged capillaries. The congested appearance of the inner membranes of the brain, particularly in the occipital region, is sometimes due to gravitation of blood to these parts either before or after death. In hyperemia of the brain an excessive amount of blood is retained within the capillaries, just as in congestion of the face or any visible portion of the body the abnormal condition is chiefly one in these vessels. In active or fluxionary hyperemia too much blood is driven from the arteries into these capillaries, while in a static or passive hyperemia the flow from them is obstructed.

Diagnosis.—The diagnosis of acute fluxionary hyperemia of the brain and its inner membranes may in some instances be made from the appearances presented by the patient, and yet in a large experience I have seen only one or two cases in which this could be done with certainty. In one of these cases the patient was relieved from threatened catastrophe by prompt and free bloodletting. He complained of severe headache, throbbing, dizziness, and a sense of peril; his face was flushed to turgescence, his heart action was rapid and thumping, and his pulse was full and tense. This patient was of plethoric habit and addicted to the moderate use of alcohol. Similar evidences of active hyperemia are occasionally observed in cases of focal lesion. The diagnosis of local hyperemia of the brain is often made in chronic cases, and is frequently erroneous. Patients suffering from all sorts of head pains and paresthesias are said to be the victims of chronic hyperemia of the base or other part of the brain; but such cases bear other interpretations. They may be mild toxemias, or light forms of neuritis of branches of the trigeminal, or perhaps they are instances of neural rather than of cerebral congestion. They may also be simply hysterical, hypochondriacal, or neurasthenic manifestations. The diagnosis of encephalic hyperemia from serious organic focal affections, such as aneurism, embolism, thrombosis, and tumor, is usually readily made by the greater intensity of the symptoms in the latter cases, and by their more focal character. No form of *persisting* paralysis, spasm, anesthesia, or disorder of the special senses can be justly set down to hyperemia, even admitting that it may at times be local; although *temporary* aphasias and paretic states may perhaps occasionally be due to local hyperemia of obstructive origin. In uremic attacks the appearance of the patient, his temperature, pulse, and respiration, and the presence of albumen and casts and other evidences of renal disease, are sufficient to make the diagnosis from congestion.

Prognosis.—As a rule, the immediate prognosis of an attack of acute hyperemia of the brain is good. A congestive attack of the apoplectic type may result fatally, capillary or arteriole extravasation

taking place in some vital region, like the floor of the fourth ventricle. Under appropriate treatment, most of the fluxionary cases of hyperemia recover, although it may be to have succeeding and more serious attacks. In chronic hyperemia with recurring exacerbations, changes gradually take place in the arachnopia and cortex, so that after a time the case is one of chronic hyperemia plus certain organic degenerative lesions. The soft membranes gradually become more or less opaque, and the vessels undergo changes. The prognosis of passive congestion due to obstruction is practically that of the disease causing the obstruction, although special measures may temporarily relieve the hyperemia.

Treatment.—Patients with a tendency to active congestion of the brain should carefully regulate their lives so as to avoid the conditions predisposing to such attacks. They should give close attention to the proper regulation of the gastrointestinal tract, should avoid stimulants, their diet should not be too luxurious, and they should take sufficient exercise in the open air. The use of the alkaline and cathartic mineral waters, natural or artificial, has a place in the treatment of such cases. When an attack of hyperemia is threatened and its coming is announced by tinnitus, throbbing sensations, flushings, and other phenomena, much can be done towards aborting or rendering less serious the attack by saline and mercurial purgation, complete rest with elevation of the head, and the use of hot foot baths, and of sinapisms and other derivatives to the extremities. If the patient has an overacting or hypertrophied heart, aconite or veratrum may be used, and the bromides and hydrobromic acid are of especial value in this form of hyperemia. Ergot can be administered with advantage unless the attacks occur at the menstrual period or for some other reason it is contraindicated. Venesection, leeches to the temples and mastoid, the application of cold to the head, and the use of enemata are old but useful measures in well chosen cases. As a rule, it is not well to treat the paroxysms of hyperemia which occur in chronic alcoholism and in paretic dementia by general bloodletting. In many cases the treatment will be mainly that of the diseases from which the hyperemia originates; for example, the use of remedies to regulate a diseased heart; or the treatment of an obstructing tumor within the cranium, or in the neck or the mediastinum. The underlying condition in many cases of encephalic hyperemia is one of toxemia; and venesection, purgation, or the use of diuretics and diaphoretics may relieve the toxic state, as well as unload the vessels; but in some cases benefit will be derived from the use of remedies directly designed to attack the acute or chronic toxemia. In such cases, for example, sodium salicylate, lithium preparations, or colchicum may be advantageously combined with the bromides, or the potassium salts may be used for their known powers of changing the quality of the blood.

ANEMIA OF THE BRAIN AND ITS SOFT MEMBRANES.

Definition and Varieties.—In cerebral anemia, strictly speaking, the amount of blood in the vessels of the brain and in the vessels of the closely investing arachnopia is proportionately less than in other parts of the body; but, as a rule, the affection is a part of a general condition of anemia and malnutrition. The diagnosis of anemia of the brain and its soft membranes has often been incorrectly made. The cerebral capillaries may contain a deficient supply of blood, just as in hyperemia an excess of blood may be present. Encephalic anemia may be acute or chronic, general or partial. A change in the quality of the blood will affect the entire brain, while a deficiency in the quantity may affect either the whole or a part only. (Gowers.)

Symptomatology.—In acute general anemia of the brain the patient is at first dull or drowsy, and may have tinnitus, sensations of confusion, or slight vertigo with feelings of general weakness. The face and conjunctiva are often pallid. If the anemia is extreme, syncope ensues, and the patient falls and remains for a short time unconscious, the skin being cool and moist. The severest forms of acute cerebral anemia are attended with convulsions; and in very rare cases, as after large hemorrhages, the patient passes into a comatose state and dies. In acute recurring cerebral anemia the patient recovering from one attack is comparatively well, but suffers from more or less headache and discomfort in the head. In a short time another and soon still another acute attack may occur. In chronic general anemia the patient usually suffers from dull headache, incapacity for cerebral work, mental depression, and irritability. Relief is afforded by the horizontal position in chronic as in acute anemia. The horizontal position favors the afflux of blood towards the brain, while the vertical position has the opposite effect. Verticality improves cerebral hyperemia, while the reverse is true of anemia. The symptomatology of partial anemia of the brain is a part of the symptomatology of those focal diseases of which it may be one stage, as of embolism, thrombosis, tumor, or meningitis. It will be especially considered when treating of embolism. Marshall Hall first recognized an acute form of anemia occurring in infants, to which he gave the name of hydrocephaloid disease. As already stated, the symptomatology of this disease bears some resemblance to that of marantic thrombosis. The little patients, usually after some exhausting disease, as an attack of diarrhea or cholera infantum, become stupid or somnolent. The face is pale. In some cases strabismus, contracted pupils, and rigidity of the neck are present. The fontanelles are usually depressed. The patient may pass into a comatose state and die, or may recover under appropriate treatment.

Etiology.—Acute encephalic anemia is sometimes of abrupt origin, as when it results from an injury to a bloodvessel, from

hemorrhoids, excessive menstruation, loss of blood during labor, or a profuse diarrhea. It may also be induced by sudden emotion, as when fright interfering with the action of the heart causes syncope. It may result from compression. Some drugs produce either temporary or persistent anemia of the brain. Roy and Sherrington found that chloral produced contraction of the vessels of the brain, which came on gradually and persisted for some time, while alkalies in general caused prompt and decided anemia. Partial anemia of the brain occurs as the result of closure or partial closure of the vessels going to certain brain areas, as in syphilis, and in tubercular and senile inflammations and degenerations. The vascular tubes become more and more narrowed. Complete closure of the artery, as in embolism and thrombosis, may at first cause extreme anemia and later necrosis of the tissue in part of the anemic area. Vasomotor spasm is still another cause of partial anemia of the brain.

Pathological Anatomy.—The anemic brain looks as if its vessels had been thoroughly washed out with water, the medullary substance presenting a dead white appearance which contrasts strongly with that of the gray matter. As a rule, it is of firm consistence. It is important to keep in mind the fact that the appearances of the brain after death are not always indicative of the antemortem conditions, a remark which has peculiar force in connection with the study of hyperemia and anemia. Occasionally the brain substance is edematous, and in cases of chronic anemia microscopical examination shows degenerated vessels and nerve cells.

Diagnosis.—Ordinarily the diagnosis of cerebral anemia is not difficult. It will be indicated by the manner of onset, the feelings of the patient, the state of the pulse, the pallor of the face, and the history of a sufficient cause. Occasionally, especially in subacute and chronic cases, the most important diagnosis is between cerebral anemia and hyperemia. Headache, for instance, is usually more diffuse in hyperemia, while in anemia it is more likely to be localized, especially about the vertex. The mental disturbances in anemia take the form of incapacity to fix the attention or perform any work requiring intellectual exertion; while in hyperemia psychical symptoms of a distinct and striking character, such as hallucinations and great excitement, may be present. Contraction of the pupils is usually indicative of hyperemia, and dilatation of anemia. In the latter the urine is usually limpid and passed in great quantities; while in hyperemia it is, as a rule, not increased and is often loaded with phosphates and urates.

Prognosis.—The prognosis of acute encephalic anemia is generally favorable, unless its cause cannot be controlled, as when a hemorrhage or a profuse discharge cannot be stopped. When chronic anemia is not associated with organic disease, and when the life and habits of the patient can be properly directed, the prognosis is good.

The form of anemia in children known as hydrocephaloid disease, if recognized early, is amenable to treatment.

Treatment.—When anemia is systemic, as it is in most chronic cases, the treatment is that of general anemia,—good food, iron and arsenic, and small doses of mercury to improve the quality of the blood. Arsenic is best given in Fowler's solution in increasing doses, as a solution of chloride of arsenic, or as arsenous acid in pills. An efficient preparation is one of the "three chloride" or "four chloride" mixtures now so generally in use. In acute cases the patient should at once be placed in a recumbent position with the head low and stimulants should be administered. For syncope, ammonia or nitrite of amyl to the nostrils, cold affusions and mustard to the spine, and in extreme cases hypodermatic injections of strychnine and digitalis, will be useful. In severe cases transfusion may be employed. Both in acute and in chronic anemia of the brain, nitroglycerin may be of value; opium and cannabis indica are other remedies which have a deserved reputation. The Weir Mitchell rest treatment thoroughly carried out, with massage, faradism, full feeding, and the use of iron, arsenic, and mineral tonics, is often most efficient. When anemia of the brain is due to cardiac weakness or disease of the valves of the heart, one of the first indications is to regulate the circulation by the use of such remedies as digitalis, spartein, strophanthus, cactus grandiflorus, and in some cases preparations of ammonia and alcohol.

EDEMA OF THE BRAIN.

Edema of the brain is an effusion of serous fluid into or beneath the encephalic membranes, or into the substance of the brain. Formerly it was customary to discuss it as a separate disease; but in recent years the tendency has been to consider it only in connection with those systemic diseases and focal disturbances of which it is a feature. Its occurrence can, I believe, occasionally be recognized by a certain array of symptoms, although its diagnosis is not, as a rule, easy. Preston concludes (1) that it should receive recognition both from a clinical and from a pathological standpoint; (2) that it follows the laws of edema elsewhere in the body, with the important exception that these laws must be considerably modified by the anatomical arrangement of the lymph spaces of the brain and its membranes; (3) that the effused serum may exert injurious mechanical pressure and also offer occasions for toxic influences; (4) that it would be a more common and serious affection were it not for the free communication which exists between the various lymph spaces, as shown by the decided symptoms produced when these cavities are isolated by inflammatory adhesions. The anatomy of the lymphatic nervous system, which it is necessary to understand in order to comprehend the mechanism of cerebral

edema, has already been sufficiently considered (pages 98 and 99). The ventricles are great lymph spaces, and many other spaces for this fluid are found in and beneath the membranes of the brain, around its vessels, and even around its cells. Numerous as are these spaces and greatly as they differ in size, they are all in some way connected with one another,—and not only with one another, but with the intracranial sinuses and with the neural and spinal lymph spaces. One important function of this extraordinary lymph system is to equalize brain pressure when overdistention of the vessels takes place.

Varieties.—Edema of the brain may be *partial* or *general*, *acute* or *chronic*. Partial edema is the result of local inflammation, or of pressure from a focal lesion, as a tumor or an aneurism. In this way may occur obliteration of lymph channels, or lymphatic or venous obstruction which leads to vascular overdistention and serous effusion in limited areas. General edema of the brain and of its membranes results most frequently from causes which affect the entire system, as from infectious diseases or diseases which lead to toxemias, or from the introduction of toxic agents into the blood. Edema is spoken of as *inflammatory* when it accompanies the extreme hyperemia which is a part of local inflammation. It may be found associated with diapedesis at the periphery of focal lesions. Varieties of edema of the brain based upon etiology—as the congestive, uremic, or infectious—might be described. Practically all forms of edema of the brain may be called secondary, as they are nearly always dependent upon systemic or special local conditions. The nearest approach to a primary cerebral edema would be an intracranial effusion dependent upon deficient vasomotor tone of unknown origin.

Clinical History.—The symptomatology of cerebral edema will be commingled with that of the disease which it accompanies or of which it forms a part; but certain symptoms are due to the edema, and it is with these alone that we are here concerned. The recognition that these are due to the edema may temporarily guide treatment. If the effusion is general and infiltrating, the symptoms will naturally be those of pressure and of irritation, the latter being due largely to distention. The intelligence is obtunded suddenly, rapidly, or slowly, according to the manner of occurrence of the edema. The patient's condition may vary from slight obtuseness to sopor, stupor, or coma. Occasionally general convulsions, agitation, or even delirium, are present. Hyperesthesia and a condition of general weakness are common. Twitchings and rigidity, with contractures, are occasionally noted. The structural stretching and strain to which the brain tissues are subjected in cases of acute or rapid origin account for these and other symptoms of irritation. Overdistention of the iter and of the fourth ventricle may give rise to cardiac, re-

spiratory, and cranioneural symptoms. The old view that in some cases of cerebral edema death occurs as the result of the compression or squeezing to which the brain is subjected is probably correct. In these cases additional symptoms may be general muscular relaxation, paralysis of the sphincters, and abolition or profound alteration of the reflexes. The edema which accompanies senile wasting of the brain, or the local atrophy which occurs in paralytic dementia and chronic epileptic insanity, does not give a symptomatology which can be separated from that of the disease of which it forms a part. Edema of the brain occurring in the course of Bright's disease and of various dyscrasias may result in death, but more frequently it will recur a number of times before a fatal issue. Obviously nothing can be dogmatically stated as to its duration, which necessarily depends upon a variety of matters, as the strength of the patient, the activity of the treatment, the continuance of the cause, and the extent of the effusion.

Etiology and Pathogenesis.—Mechanical theories account for the production of encephalic edema in some cases, but not in all. Preston found that clamping or tying one or both jugular veins was not followed by an intracranial edema the evidences of which were present after the death of the animal. Embarrassment of the circulation and respiration, and convulsions and spasmodic twitchings, were present. He concluded that simple constriction of the venous circulation is sufficient to cause lasting cerebral edema. Other causes of edema of the brain are alterations in the blood, changes in the vessels, and faulty innervation. These factors must be unusually active or the effusion must be unusually great in order to cause persistent edema. The most powerful of these factors is a disturbance of the lymph system. Diseases accompanied by an excess of venous tension, as chronic affections of the heart and lungs, may cause edema, especially when the broken down compensation throws into the cellular tissue of the viscera and of the splanchnic cavities the overflow of the venous system. Sometimes the circulatory disturbance is entirely local and provoked by an obstacle in the network of the cerebral veins, and above all at their most important point, the vein of Galen ; this may be caused by a tumor of the brain, a focus of meningitis, or a neoplasm of the neck or of the mediastinum. (Grasset and Rauzier.)

Pathology and Morbid Anatomy.—During the postmortem examination of an edematous brain much of the fluid usually escapes, but when the autopsy is carefully made a bluish dropsical appearance of the arachnopia is seen over larger or smaller areas of the cerebral surface. If the brain has been infiltrated with serum, it is usually pale and soggy, a "wet brain ;" but in old cases of subarachnoid edema even the opposite of this may be the case, the convolutions being flattened and separated. On section of the brain substance

often a small quantity of serous fluid runs out. The ventricular liquid is increased. The microscope shows dilatation and distention of the perivascular spaces. These lesions differ so little from the phenomena of cadaveric maceration, except in the abundant exudation, that certain authors have attributed this lesion of cerebral edema to a postmortem infiltration of the intraventricular liquid into the cerebral tissue. A circumscribed area compressed by the edema is occasionally softened. (Grasset and Rauzier.)

Diagnosis and Prognosis.—The diagnosis of cerebral edema in a given case will be made largely by a knowledge of the disease of which it is an accompaniment or a result. When unconsciousness, with or without convulsions, comes on in the course of chronic nephritis, cerebral edema may be suspected. The determination of lesions which might cause venous obstruction would point to this diagnosis. Aside from the previous history of the patient and of concomitant disease, the diagnosis must be largely made by the exclusion of such affections as encephalic hemorrhage or softening, sinus thrombosis, and toxic insensibility. The prognosis as to immediate results is uncertain. The patient often dies ; but if he recovers from the attack it is in the majority of cases only to have succeeding attacks. The real prognosis is dependent upon the nature and severity of the chronic affection which gives rise to the edema.

Treatment.—The treatment should be in part to improve the general condition of the patient and favorably affect the chronic disease from which he is suffering, and in part to relieve immediately the threatening symptoms. In some cases the use of the hot pack, and that of jaborandi internally, or of pilocarpin by hypodermatic injection, may be indicated, or other forms of diaphoretic treatment may be employed. Cathartics and diuretics are of service on the same principles that guide their use in chronic renal diseases with effusion anywhere. Arsenic, iron, and other tonic and constructive remedies are of importance in the continuous treatment of the patients.

DISEASES OF THE WALLS OF THE ENCEPHALIC BLOOD-VESSELS.

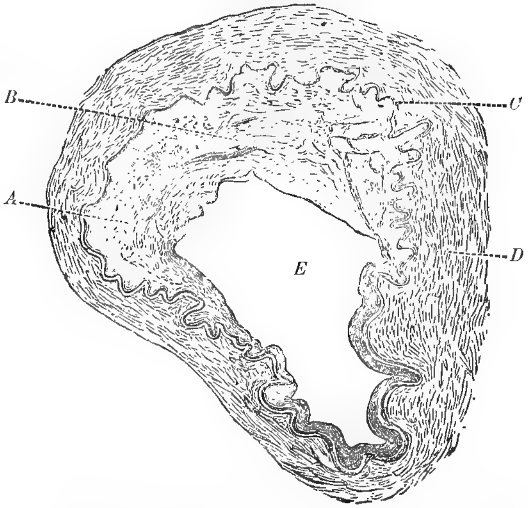
Diseases of the walls of the encephalic vessels and derangements of the circulation require separate consideration, although the latter are often dependent upon the former. Diseases of the vessel walls should be considered first, because they lead to obstructions and derangements of the circulation. Widespread disease of the encephalic bloodvessels may interfere to such an extent with the tone and nutrition of a portion of the entire brain as to give symptom pictures which deserve a nosological place. Before taking up these affections it may be well to recall briefly the constitution of the vessel walls. Arteries are usually regarded as constituted of either three or four

coats, which can be readily distinguished in a cross section of an encephalic artery of moderate size. When three tunics are enumerated they are described as the *tunica adventitia*, *tunica media*, and *tunica intima*. When a fourth coat is recognized it is usually made by a subdivision of the intima or inner tunic into an *elastic* and an *endothelial layer*. The muscular coat is poorly developed in the larger arteries. The walls of the veins have coats like those of the arteries, but they are somewhat thinner, especially the muscular coat. The capillaries are lacking in the muscular coat, appearing to be simply continuations of the endothelial layer, but they sometimes have an areolar investment.

Varieties of Arteritis.—The chief varieties of inflammation of the arteries of the brain are the *atheromatous*, the *obliterative*, *aneurismal periarteritis*, *nodose periarteritis*, and *acute purulent inflammation*. Warty or varicose endarteritis, in which excrescences form in the vessels, rarely attacks the arteries of the brain. The so-called malignant endarteritis, which has the same character as ulcerative endocarditis, has some importance in encephalic pathology, because the vegetations which form in the cranial vessels may be carried into the endocranial circulation, although the disease rarely originates in the brain. In rare instances it attacks the large encephalic vessels. Atheromatous arteritis, the usual predisposing cause of aneurism, is primarily a disease of the inner coats of the bloodvessels. In atheroma the intima first presents a milky opacity in patches, which become elevations, and in the midst of which appear pale yellow spots, visible from the outside. They either ulcerate or calcify. The ulcerations predispose to calcification and aneurismal dilatation, an important fact in connection with cerebral hemorrhage. When they calcify without ulceration the patches form *calcareous scales*, which are liable to be dissevered from their attachments and to be carried off in the blood stream to become emboli. The arteries of the circle of Willis are especially prone to atheroma. Atheroma is sometimes due to syphilis, and probably also to gout and rheumatism, and is frequently associated with disease of the kidneys. It is an accompaniment of old age and of premature senility. The intima sometimes shows spots of fatty degeneration instead of the atheromatous patches just described. The arterial coats become inflamed, pus forming between them, and cerebral abscesses may be developed around the arteries. Charcot in particular has described a form of periarteritis which especially affects the vessels of the brain and may lead to miliary aneurisms. The disease usually progresses from without inward, the inner coat being seldom affected. It is most marked in small arteries, and the capillaries may be attacked. Syphilitic inflammation of the arteries presents itself in the form of either an endarteritis or a periarteritis, and both may be present in the same case. Syphilitic endarteritis is usually of the obliterative type.

Our first exact knowledge of it was largely due to Heubner. It is a matter of radical importance to remember that, while atheromatous endarteritis leaves the walls of the vessels unchanged in thickness and leads to fatty degeneration, syphilitic endarteritis augments their thickness. In time even a comparatively large vessel may become obliterated as the result of gradual encroachment upon its lumen by syphilitic endarteritis. In Fig. 270 is shown an illustration of endarteritis of the basilar artery. Necrotic softening is a frequent result of obliterative endarteritis, and this, because of the diffusion of the process, is likely to be multiple. In some cases the parts may be imperfectly supplied with pabulum through collateral circulation, and hence a deteriorated but not necessarily a softened tissue is the result.

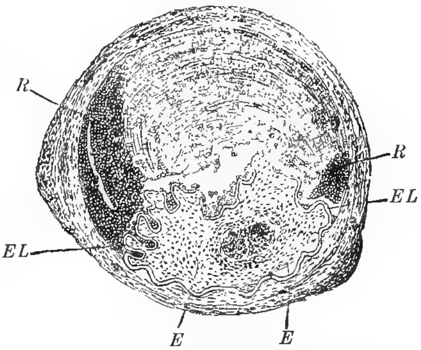
FIG. 270.



Syphilitic obliterative endarteritis of the basilar artery: *E*, lumen, about two thirds obliterated; *D*, the muscular coat and adventitia nodulated by inflammatory changes; *C*, the elastic lamina; *A*, inflammatory new formed tissue in the intima, composed of large spindle and round (on section) cells, with normal cells of the intima next to the elastic lamina and the lumen of the vessel; *B*, the hyaline substance seen distinctly with a higher power. (Weber.)

Another result of syphilitic endarteritis, especially in the brain vessels of medium or large size, is the production of aneurisms, which in their turn give rise to intracranial hemorrhage. Next to embolism it is the most frequent cause of aneurisms. In Fig. 271 is shown syphilitic endarteritis of the medicerebral artery with a dissecting aneurism. All cases of massive hemorrhage into the brain substance associated with syphilitic endarteritis are not, however, the direct results of aneurismal rupture. Several varieties of syphilitic periarteritis are well

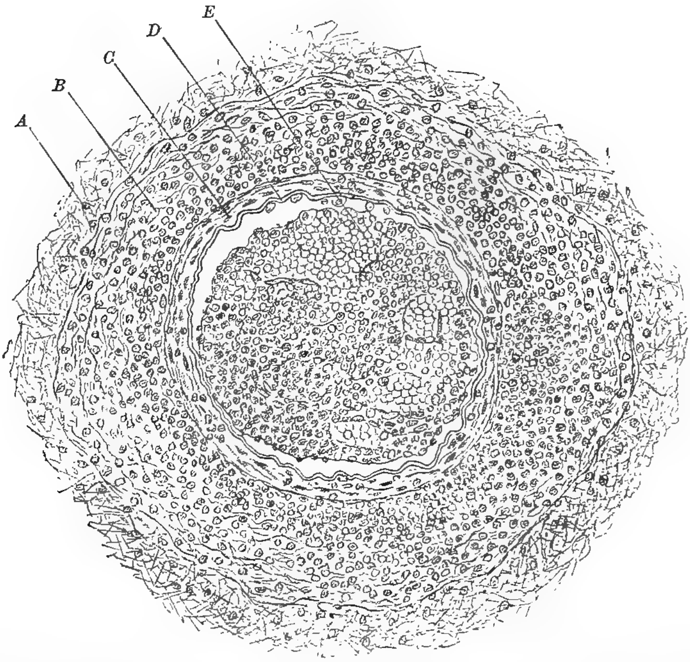
FIG. 271.



Section showing endarteritis and a dissecting aneurism of a branch of the left medicerebral artery: *R*, *R*, blood cells heaped together; *EL*, elastic layer; *E*, *E*, intima. (Herter.)

known. One of the most important of these is nodose periarteritis, which has been described by Kussmaul, Meyer, Bruce, and others. In this affection irregular infiltration of the outer sheath of the vessel occurs. In Fig. 272 is shown in section the appearance of a vessel affected with one of the forms of periarteritis with an organizing thrombus. One form of nodular periarteritis is not due to syphilis, but the recorded cases show that the syphilitic variety attacks the arteries of the brain with great frequency, while just the reverse is true of the nonsyphilitic form, the brain usually escaping although

FIG. 272.



Microscopical section through a vessel in a case of cerebral syphilis, showing great infiltration and thickening of the outer coat, and an organizing thrombus in its interior: *A*, inflammatory products around vessel; *B*, thickened outer coat; *C*, middle coat; *D*, inner coat; *E*, organized thrombus containing delicate new formed vessels in its interior. (Bramwell.)

many organs may be affected. Syphilitic nodose periarteritis attacks particularly the great vessels of the base and their branches; it may or it may not be associated with obliterative endarteritis. A most important fact for the clinician to bear in mind is that "very frequently vascular changes are associated with a diffuse basal meningomyelitis with foci of softening in the nerve centres, and interstitial cellular infiltrations of the cranial nerves." (Bruce.) These cases of nodose periarteritis may show infiltration of both epineurium and endoneurium. In one form of syphilitic periarteritis the disease chiefly attacks the minute vessels, and results in sclerosis and atrophy.

Porter has described a form of hyaline degeneration due to syphilis, the result of a deposit of proteid substance at first in the walls of the smaller arteries and arteriocapillaries and finally throughout the arterial system. This deposit weakens the walls of the vessel, the lumen of which is sometimes contracted and sometimes expanded. Typical miliary aneurisms arise when this thinning and expansion are sharply defined and localized in the smaller vessels.

Chronic Endarteritis (Arterial or Arteriocapillary Fibrosis or Sclerosis).—Gull and Sutton in 1872 and again in 1876 and 1877 called attention to a pathological condition of the capillaries, arterioles, and interstitial structures of various organs, such as the kidneys, heart, spleen, brain, spinal cord, and lungs. This was described by them as fibroid degeneration. The degeneration of the bloodvessels observed by others, particularly in connection with contracted kidneys and hypertrophied heart, was regarded by Gull and Sutton as not dependent upon renal disease as had been supposed. They believed that it was rather a part of a general pathological process and one that especially attacked the vessels. According to Meigs, the name fibrosis, or rather *arteriocapillary* fibrosis, is not well chosen, because the disease is not truly a fibrosis and the capillaries are not necessarily implicated. The same objection would hold against *sclerosis.* Meigs believes that the affection is essentially a chronic endarteritis, inflammatory thickening of the arteries and arterioles being the most constant lesion. Whatever view may be taken of the pathology of this affection, the student of nervous diseases must early learn to recognize its importance, keeping in mind that it is not only a factor in the production of many focal diseases, but may also present a symptomatology of its own, which may be referable to the brain or to other organs. The affection described by Gull and Sutton and by Meigs, and the hyaline degeneration of Oeller, Porter, and others, may represent differing phases of the same general process, or perhaps somewhat differing morbid processes which lead to similar results in the vessels and viscera. Chronic endarteritis begins in the intima as a simple inflammation, and may or may not extend to the veins and capillaries. While it shows a tendency to invade vessels everywhere, it may choose certain organs for its early inroads or to bear the brunt of its assaults. The brain vessels in particular suffer, giving an array of distinctive symptoms. These symptoms cause frequent mistakes in diagnosis. Neurasthenia, hysteria, chronic meningitis, and brain tumor are some of the affections which may be wrongly supposed to exist. Meigs found chronic endarteritis typically present in a marasmic child who died at the age of five months, and it has been observed and studied at all periods from early infancy to extreme old age, although most frequently it is a disease of advanced years. When cerebral hemorrhage occurs in infancy, childhood, or youth, its presence may with good reason be suspected.

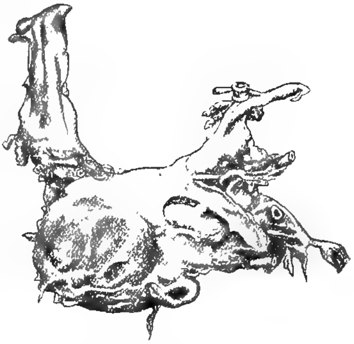
Symptomatology of Encephalic Endarteritis.—Among the symptoms of chronic endarteritis are general muscular weakness, shuffling gait, wasting of the muscles, loss of memory, change of disposition, and mental irritability. Meigs has noted a curious hyperesthesia in old people, not confined to the skin, but caused by movement of any part of the body. Epistaxis, edema of the ankles and feet, albumen and casts in the urine, marked irregularity of the cardiac valves, and lung symptoms such as bronchitis, with or without expectoration, may be present. The pulmonary symptoms may come on abruptly with fever or may develop insidiously. One of the most distressing symptoms of chronic endarteritis or arterial sclerosis is *vertigo.* Grasset has divided the vertigo which occurs in these cases into simple vertigo, vertigo with epileptiform crises, and vertigo with slow pulse and syncopal epileptiform attacks, the terms descriptive of these variations indicating the special characteristics of each. The vertigo usually first presents itself as an attack of sudden faintness or swimming in the head, a feeling of giddiness or of distinct gyration, or of darkness and impending death. The attacks occur at intervals and vary considerably. Some cases experience a sense of fulness or throbbing in the head, a feeling of heat in the scalp, and blurring of vision. The face may be at first pale and later flushed, and the patient may have a strong desire to get into the open air. When the vertigo is severe he may sink, stagger, or fall to the ground, but consciousness is rarely lost. Careful examination of these cases will generally show a tortuous temporal artery, a stiffened radial, a distinct arcus senilis, a strong or even clanging sound of the heart, increased arterial tension, a pulse slow or arhythmic, and scant urine with a trace of albumen. Exertion or change of position from lying or sitting to standing may cause the attacks. Ménière's disease, or at least aural vertigo, may be wrongly diagnosticated, especially if some tinnitus or impairment of hearing is present. Recognized in an early stage, potassium iodide, sodium iodide, or hydriodic acid may prove of some service in retarding the progress of the disease. The treatment should be continued over a long period. Arsenic and alkaline mineral waters are advantageously combined with the iodides.

ENCEPHALIC ANEURISM.

Varieties.—Aneurism of the brain, as of other parts of the body, may be *true* or *false.* The former is constituted by the coats of the vessel in which it originates; the latter is an enlargement due to the condensation and accumulation of neighboring tissues. Cerebral aneurisms are usually fusiform or saccular. A *varix* is an aggregation of small adjacent dilated arteries. A *varicose aneurism* or *arteriovenous aneurism* results from the adhesion of an arterial aneurism to a vein, a communication becoming established between

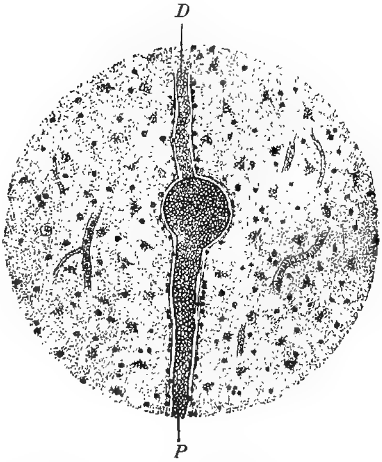
the two. The seat of encephalic aneurisms is in the first place usually in the large vessels of the base or in their immediate branchings. The illustration shown in Fig. 273 is of a large aneurism of the medicerebral artery from a specimen in the Pathological Museum of the Philadelphia Hospital. In such an aneurism, symptoms referable to the effects produced by the enlargement upon neighboring parts with known function are present; in other words, the aneurisms act as tumors, giving rise to focal cerebral symptoms. Osler found twelve cases of cerebral aneurisms in eight hundred autopsies at the Montreal General Hospital; but this was an unusually large proportion. The medicerebral artery is the most frequent seat, the basilar, internal carotid, and precerebral come next, and other of the medium and large vessels are affected with less frequency. Secondly, they may be of small vessels and may show their effects only in symptoms which indicate disturbance of the circulation. These minute aneurisms are of importance in the etiology of special focal diseases, and particularly of cerebral hemorrhage. This form of aneurism was first elucidated by Charcot and Bouchard, although previously described by Virchow, and probably by Cruveilhier. Miliary aneurisms are sometimes found in large numbers in a single brain; a few perhaps are seen at the base, but they are abundantly present in the vessels that dip into the fissures, course over the crests of the convolutions, or penetrate into the centrum ovale, capsules, and ganglia. A cortical aneurism is shown in section in Fig. 274. As already indicated, these aneurisms are due to a form of periarteritis.

Fig. 273.



Aneurism of the medicerebral artery.

Fig. 274.

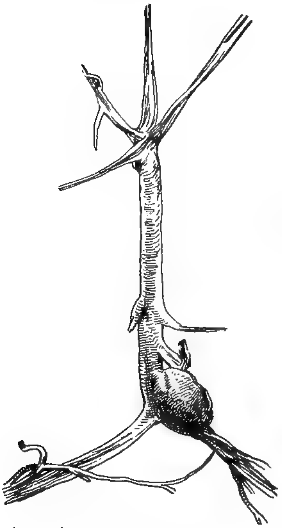


Miliary aneurism in the cortical substance of the brain, divided longitudinally: *P*, proximal side of the vessel, its diameter being $\frac{1}{54}$ of an inch; *D*, distal end of the vessel, its diameter being $\frac{1}{128}$ of an inch, the greatest diameter of the aneurism being $\frac{1}{20}$ of an inch, its smallest diameter $\frac{1}{29}$ of an inch. (Porter.)

Symptomatology.—With the addition of a few special phenomena, the general symptoms of intracranial aneurisms are the

same as those of small tumors. Sensations of pulsation and throbbing in the head are supposed to indicate the existence of an intracranial aneurism, and this may occasionally be true; but usually such feelings are due to other causes, as general nervousness, cardiac irregularities, or vasomotor disorders. A bruit caused by a vessel within the skull is very difficult and usually impossible of determination. A systolic brain murmur heard in the temporal region may be wrongly supposed to be due to an aneurism, and physicians show an unusual tendency to deceive themselves as to the presence of a bruit from extracranial auscultation. Pain in the head may be caused by an encephalic aneurism, but it is not of the same intensity as that produced by growing neoplasms. Optic neuritis is of rare occurrence. The focal symptoms of encephalic aneurism are of a decided character only when the dilatation is considerable. They may be entirely wanting. Basilar aneurisms have been reported without characteristic symptoms. The basilar aneurism shown in Fig. 275 is from a case recorded by Porter. The patient had a history of syphilis, and of a fall eight years before his death. Three months before his death he suffered from severe headache and pains in the cervical and occipital regions, with melancholia and mental inertia. Other symptoms noted a few days before death were convulsions, facial twitchings, slight photophobia, contracted pupils, and slight ptosis. Occipital neuralgia may be a symptom of aneurism of a vertebral artery. Oppenheim has recorded a case of vertebral aneurism due to atheroma which caused structural changes in the postoblongata in the neighborhood of the vagus nucleus and thus gave rise to grave disturbances of respiration. The third nerve lies between the postcerebral and precerebellar arteries, so that an aneurism or nodose periarteritis of these vessels might compress this nerve and cause oculomotor palsy. A pulsating exophthalmus is sometimes caused by an aneurism of the internal carotid in the cavernous sinus. An aneurism of the internal carotid may so compress the chiasm as to cause a bitemporal hemianopsia, as in a case recorded by Mitchell and Dercum. In aneurisms of the arteries of the base, epileptiform convulsions and psychical disorders have been noted. In Fig. 276 is shown an aneurism of the medicerebral artery from a case recorded and illustrated by Blackburn in the Report of

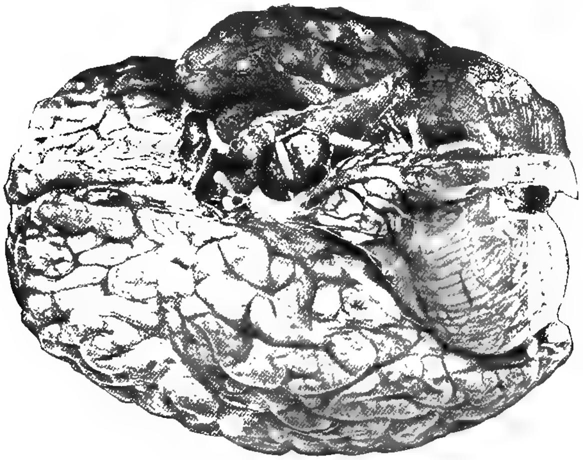
Fig. 275.



Aneurism of the vertebral and basilar arteries, showing its exact position and size after the separation of the vessels from the base of the brain. (Porter.)

the Government Hospital for the Insane for 1894. This patient was sixty-four years old, and had convulsive attacks occasionally which were believed to be epileptic; he was somewhat demented, was feeble and kept in bed, but showed no distinct paralysis except ptosis of

FIG. 276.



Aneurism of the medicerebral artery. (Blackburn.)

the right eyelid. He had occasional attacks of vomiting, and a few days before he died the vomitus contained blood. The eyes were not examined with the ophthalmoscope, but sight was impaired. The duration of his mental trouble was over eighteen years. Death occurred in a convulsion.

Etiology and Pathology.—One of the most common causes of cerebral aneurism is endarteritis, which is usually of syphilitic origin, but aneurism occurs with considerable frequency in nonsyphilitic atheromatous vessels, and it may also result from other forms of vasal disease. Sometimes it is of embolic origin, dilatation occurring as the result of the changes in the arterial walls at the site of the embolism. Aneurism of the intracranial vessels is frequently associated with endocarditis. Encephalic aneurism may occur at any age in syphilitic subjects; in others it is more frequent in middle life. In rare instances it has been noted in very young children. Males show a greater liability than females to intracranial as well as to other forms of aneurism.

Diagnosis.—The diagnosis of encephalic aneurism is nearly always difficult. It can be made with an approach to certainty only by the detection of a bruit, when the conditions which predispose to aneurism, such as a history of syphilis or of cardiac and vascular disease, are present. Probably the only arteries which give a bruit that can be determined by auscultation are the carotids, vertebrals, and basilar, and even the last is doubtful. With Dr. J. B. Deaver,

of Philadelphia, I saw a case in which the diagnosis of arteriovenous aneurism was made chiefly because of the presence of a tremor similar to that caused by such aneurisms, because also of the presence of a bruit, and because pressure on the carotid arteries seemed to lessen the pulsation and bruit. The symptoms were chiefly those of a cerebellar growth, which was one of the diagnoses made, but a pulsating tumor was present and protruded through a small opening in the skull to the left of the occipital protuberance. The autopsy showed a gliomatous tumor which had occupied much of the fourth ventricle, reaching into the middle lobe of the cerebellum and both cerebellar hemispheres. The ventricles of the brain and their horns were enormously dilated. When the spinal cord was severed from the oblongata, the spinal canal was found to be one sixth of an inch in diameter, and from it much fluid escaped. The case was therefore one of gliomatosis with hydrocephalus and hydrorrhachis, the opening in the skull having apparently taken place as the result of the enlargement of the brain causing continuous pressure and erosion of a thin skull. Next to a carotid or a vertebral bruit, the symptoms most significant of aneurism are those which point to pressure or invasion of certain cranial nerves and of special regions of the base. Unless an aneurism has attained considerable size, focal symptoms are not distinctive. The diagnosis is to be made from such affections as thrombosis of the cavernous sinus, basal tumor, and meningitis. As an aneurism of sufficient size to cause focal symptoms is in fact a tumor, the obstacles to a clear opinion are sometimes insuperable. An opinion will usually be reached chiefly by a discussion of the causes which might lead to aneurism on the one hand or to thrombosis, tumor, or meningitis on the other. The greater diffusiveness and irregularity of the symptoms in basilar meningitis will be of assistance in making the diagnosis.

Prognosis.—The prognosis of an intracranial aneurism is always grave. Not every case results fatally. The aneurism occasionally disappears spontaneously, and in rare instances coagulation is brought about either by the use of iodides or by ligature; but rupture takes place in a large majority of cases of aneurism of large or moderate size. Miliary aneurisms are well known to be among the commonest predisposing pathological causes of cerebral hemorrhage. The prognosis as to time is uncertain. Most cases last a few years, and some have a duration of many years.

Treatment.—When the diagnosis of aneurism of the internal carotid or medicerebral has been made with fair certainty, the external carotid can be ligatured; and tying of the vertebral can be resorted to for aneurism of either vertebral or of the basilar. Potassium iodide and sodium iodide should be given to prevent the spread of syphilitic disease in the vessels if the case is specific, and in any case to promote coagulation in the aneurism. A patient

believed to be suffering from an intracranial aneurism should use the same care and precautions as are called for in the treatment of an aneurism of any portion of the body. Alcoholic stimulants and all sources of physical and mental strain should be avoided. The bowels should be carefully regulated.

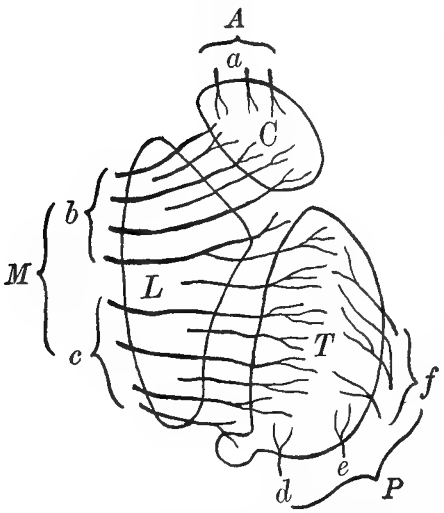
ENCEPHALIC HEMORRHAGE.

The term *apoplexy* is frequently used as synonymous with encephalic hemorrhage; but, while encephalic hemorrhages are apoplexies, not all apoplexies are encephalic hemorrhages. An apoplexy may be due to arterial embolism or thrombosis, to sinus thrombosis, to uremia or other toxemias, to acute congestion of the brain, or to the bursting of an abscess. Strictly speaking, apoplexy is a symptom, the word being derived from the Greek ἀποπλήσσειν, to strike or cripple by a stroke. It is an attack of sudden insensibility, the word "stroke" having popularly the same meaning. The most common types of apoplexy are undoubtedly due to rupture of a cerebral vessel, or to its closure by an embolus or a thrombus. Intracranial hemorrhage and acute softening from embolism and thrombosis are often considered together under the general head of apoplexy, but it is best to discuss each separately, especially as it is important and is quite possible to differentiate them, at least in their acute stages.

Varieties.—Encephalic hemorrhage in its most common forms is conveniently subdivided into (1) *superdural* or *epidural;* (2) *subdural;* (3) *cortical* or *subpial;* and (4) *intracerebral.* The first two varieties are commonly due to rupture of the meningeal arteries, chiefly the middle meningeal or medidural. They are frequently associated with fractures and other injuries of the skull, and have been considered in Chapter III. (pages 269 to 272). Cortical or subpial hemorrhage has its source in the cerebral arteries proper, most frequently in the cortical system of the medicerebral. A subdural hemorrhage may be, in its effects at least, a cortical hemorrhage as well, compressing the brain or even tearing the arachnopia and cortex. Intracerebral hemorrhage is an extravasation into the basal ganglia or the capsules, or into both, or into the white substance of any of the cerebral lobes. These hemorrhages are usually caused by rupture of the branches of the central or ganglionic system of vessels (Fig. 277), although small hemorrhages into the centrum ovale may occur from distal branches of the cortical system. Any portion of the encephalon may be assaulted by hemorrhage, so that we may have other focal varieties. The site of such hemorrhages may be the cerebellum, the ventricles, the oblongata, the pons, the crus, the quadrigeminal body, or the central region of the base. By far the most frequent type of intracerebral hemorrhage, and that on

which most descriptions are based, is that which results from rupture of the lenticulostriate artery, a fact to which reference has already been made (page 421).

FIG. 277.



Scheme of the ganglionic arteries: *C*, caudatum; *L*, lenticula; *T*, thalamus. The arteries are as follows: *A*, precerebral; *M*, medicerebral; *P*, postcerebral; *a*, anterior striate; *b*, lenticulostriate; *c*, lenticulo-optic; *f*, inferior or internal optic; *e*, posterointernal optic; *d*, posteroexternal optic. (After Testut.)

Most Common Sites of Encephalic Hemorrhage.—The most common forms, locally considered, are the capsuloganglionic, the cortical, the ventricular, those into the centrum ovale, the cerebellar, the pontile, the oblongatal, and the crural. I omit in this enumeration meningeal hemorrhages, which have already been considered. They are of comparatively frequent occurrence, especially as the results of traumatism.

Capsuloganglionic Hemorrhage.—Intracerebral hemorrhage may occur in any one of half a dozen positions with reference to the three great ganglia at the base of the brain and the internal or external capsule. With reference to hemorrhage without ventricular inundation, several locations in or near the ganglia may be diagnosticated. If the hemorrhage has occurred at a position corresponding to the knee or the anterior half of the posterior limb of the internal capsule, the chief effect is the production of motor paralysis of the opposite half of the body, with symptoms of an acute apoplectic attack. These symptoms are practically the same for all the nonventricular varieties. If it has occurred so as to be related to the posterior third of the posterior limb of the capsule where it lies chiefly between the lenticula and the thalamus, contralateral paralysis both of motion and of sensation will be the great feature. When the extreme limits of the posterior limb of the internal capsule and ganglia are the seat of extravasation, hemianesthesia without hemiplegia will be present; but this variety is rare.

Clinical History.—A good practical subdivision of the clinical history of a case of encephalic hemorrhage is into (1) the premonitory symptoms (sometimes absent); (2) the period of the apoplectic onset or hemorrhage; (3) the postapoplectic period; and (4) the period of persistent symptoms, or the chronic stage in cases which do not end fatally. The description which follows is chiefly applicable

to cerebral hemorrhage of considerable size. So far as the general features of the apoplectic attack are concerned, the symptoms given will answer for those produced by hemorrhage into a number of localities in the brain, as the centrum ovale, ganglia, and capsules, and the ventricles and cerebellum. Some differences in temperature, in respiratory phenomena, and in the pulse may be caused by the difference in the location of a clot; but if the hemorrhage is large, each variety, so far as the locality is concerned, will have a general likeness to the others.

Premonitory Symptoms. In not a few instances encephalic hemorrhage begins without any premonitory symptoms, although some ill health, recent or remote, has usually been present. The patient may have been a sufferer from renal, cardiac, and arterial disease with its accompaniments, such as urinary suppression, cardiac irregularity, and occasional vertigo, headache, mental irritability, and insomnia. In the main the symptoms will be those which were described when arteriocapillary fibrosis was considered. Bleeding from the nose and sensations of fulness in the head are frequent immediate antecedents. In plethoric patients the attack may be preceded by flushing of the face and throbbing of the visible vessels, with other evidences of congestion and increased blood pressure. In rare cases monochoreic or hemichoreic movements precede the stroke. A patient prior to the occurrence of apoplexy may come detailing certain symptoms, such as spells of dazing or vertigo, dull pains or peculiar sensations in the head. These sensations are often described as feelings of oppression, constriction, numbness, or heaviness. Instead of these cephalic symptoms, it may be that he complains of numbness, tingling, or weakness in arm or leg or in both. These symptoms are often accompanied by emotional conditions, introspection, apprehension, and lack of energy or decision.

The Apoplectic Onset. Encephalic hemorrhage is ushered in in a variety of ways. In the most common case the patient becomes suddenly unconscious, falling if not sitting or in a recumbent position; in another case unconsciousness may come on slowly, often with dazing, dizziness, and great mental confusion; in a third case, probably when the hemorrhage is comparatively small, the patient goes through the attack without ever having been entirely unconscious, although some perversion of consciousness usually takes place; in still another case the unconsciousness is at first transient—the patient rallies, but soon has a second or a third attack of insensibility, the last being deep and persistent. The apoplexy may be initiated by spasm, which may take the form of monospasm or of a general or a unilateral convulsion; but this mode of onset is rare. The spasm may be coincident with the unconsciousness or may soon follow it, its occurrence probably indicating a cortical motor site or a rapid ventricular extension of the hemorrhage. Immediate

examination shows paralysis of one side of the body, sometimes of motion only, or more rarely of both motion and sensation. Other phenomena often present are conjugate deviation of the head and eyes, fixed and often irregular pupils, absence of or marked changes in the reflexes, and involuntary evacuations of the urine and feces. If the temperature is at once taken it will be found to be lowered, and usually different on the two sides of the body, as will presently be especially considered. The pulse is generally rapid, tense, and full, and respiration is difficult, labored, or stertorous. In forty-seven cases Dana found the time of onset to be as follows: at night during sleep or sleeping hours, thirteen; in the morning on rising, eighteen; after five in the evening, six; and during the day, ten.

The Apoplectic Period.—For hours or days after the onset the patient remains in what is called the apoplectic state or stage. Some of the phenomena just mentioned persist, but marked differences in the state or degree of impairment of consciousness may be present. In a short time the attack may change its character for better or worse. When a patient does not succumb immediately or soon, in typical cases the symptoms at different stages assume more or less regular features as to temperature, respiration, pulse, delirium, and even paralytic and sensory phenomena. These and certain other special features of the apoplectic period, such as the position assumed by the patient, conjugate deviation, state of the pupils, early rigidity and contractures, the condition of the lungs, and the peculiarities of the urine, will now be briefly considered.

Temperature, Respiration, and Pulse. As a rule, during the early hours of the attack the temperature falls to 97° or even to 96° F., but a rise takes place in a few hours, generally for both sides of the body. This rise may be a degree or two higher on the paralyzed side. In cases soon fatal it may continue to ascend until it reaches 102°, 103° or even 104° F., sometimes sinking again just before death, but in other cases continuing to rise even after death. Dana found the rectal temperature ranging between 100° and 102° F. in cases which ended fatally the day after the stroke. When a very high terminal rise takes place, as to 105° or even to 107° F., pneumonia is often present. As a rule, in intracerebral hemorrhage the temperature on the paralyzed side is about one degree higher than on the unaffected side. In cases observed by the writer the preliminary fall of temperature described by Bourneville has often but not invariably been present. Hemorrhage which lacerates the cortex almost always gives rise to elevation of temperature. Buccal and rectal temperatures are higher than axillary temperature, but they are sometimes difficult to take. The buccal temperature may be hard to obtain owing to the position of the patient and the method of breathing, and the rectal temperature owing to involuntary evacuations. The surface temperatures of the face and limbs differ markedly in some

cases, being usually higher on the paralyzed side. Nothing of practical value has been determined with reference to pericranial temperatures. In an average case of hemorrhagic apoplexy the respiration becomes stertorous. Sometimes it is puffing, and has been called the "tobacco smoker's breathing." Only one side of the mouth may be puffed out. Cheyne-Stokes breathing is frequent, particularly in the late stage of a fatal case. Sometimes, as in cases reported by Growitz, respiratory movement and capacity are markedly diminished on the paralyzed side. This diminution is visible not only on forced respiratory movements but during tranquil breathing, and seems to consist in some cases of a delay of the inspiration and a premature cessation of the expiration. In rare instances the respiratory movement is more energetic on the paralyzed than on the unaffected side. The pulse varies greatly. Ordinarily it is full and slow at first, but later it becomes more frequent. It is sometimes distinctly arhythmic, and it may differ somewhat on the two sides.

Delirium. Occasionally the patient passes into a state of delirium, usually of a mild or muttering type. According to Hughlings Jackson, this delirium is merely a return effect upon the brain, a consequence of the imperfect working of the respiratory and other organic systems, a state analogous to that present in some cases of emphysema, bronchitis, or cervical fracture.

Pupillary Phenomena. A study of the pupils may be important in fixing the site of a hemorrhagic lesion. In severe apoplexies not infrequently both pupils are contracted, but dilatation even in these cases may take place as the case progresses. Dilatation of one pupil, as already shown (page 270), is a symptom of value in the diagnosis of dural hemorrhage, occasionally occurring on the side of the lesion.

Conjugate Deviation of the Eyes and Rotation of the Head. Both eyes may be turned to one side and slightly upward, as if looking over one or the other shoulder, the head and neck usually being rotated in the same direction. The deviation may be slight or it may be marked. Not infrequently the neck muscles on one side are rigid. The eyes are commonly motionless, but occasionally exhibit oscillation. In a recent apoplectic case probably due to a small hemorrhage into the internal capsule, moderate motor paralysis being present in the left arm and leg, the patient's eyes were strongly deviated to the right and at first were motionless. As the stupor passed away, the eyes gradually came to the front and left, as if striving to overcome some resisting but slowly yielding force, the consequence being a peculiar slight horizontal nystagmus. Conjugate deviation may occur not only from hemorrhage and other destructive and irritative lesions of the capsuloganglionic region, but also from lesions of the cortex, centrum ovale, cerebellar peduncles, and pons. It has been best studied in the first and last named regions. Cerebral and pontile lesions produce forms of conjugate

deviation which in some of their features are the reverse of each other. Swanzy in the following table shows how the character of the lesion affects, in each case, the direction of the deviation.

Cerebral lesions:	Destructive.	Eyes turned away from palsied side.
	Irritative.	Eyes turned towards convulsed side.
Pontile lesions:	Destructive.	Eyes turned towards palsied side.
	Irritative.	Eyes turned from convulsed side.

Conjugate deviation ordinarily lasts for several hours, or from one to two days: like drooping of one side of the face, it is an important diagnostic sign because it is revealed by inspection. The most positive and persistent forms of conjugate deviation are from lesions in the pons, but it is probable that the four chief forms of ocular coordination are represented above the crus, and hence may be interfered with in cerebral apoplexies. These coordinations are movements of both eyes to the right, movements of both eyes to the left, movements of both eyes downward and inward (narrowing of the pupils and contraction of the ciliary muscles, producing increased convergence and accommodation), and, finally, movements of both eyes upward and outward. (Priestley Smith.) Wernicke has recorded paralysis of upward and downward movements from lesions of the striatum and thalamus. Occasionally these different forms of ocular coordination are observed at different periods of the apoplectic attack in the same case. The lateral deviations are most evident, and therefore most likely to arrest attention.

Decubitus and Rigidity. The patient tends to assume the dorsal decubitus, and, if the hemorrhage issues fatally, to remain in the supine position without change. When consciousness is imperfectly retained, he may show some tendency to move to one side and to toss about the unparalyzed limbs. It is often advisable to put him at least partially on one side, both to relieve the stertor and to prevent the formation of bedsores. The limbs on the paralyzed side may present a condition of spasticity or early rigidity, which is especially marked when the hemorrhage is in the motor subcortex. This early rigidity sometimes takes place in the stage of so-called inflammatory reaction. In some cases it passes away; in others it merges into the condition of chronic contracture.

Miscellaneous Phenomena. In large hemorrhages both the skin reflexes and the muscle phenomena may be depressed or absent in the early hours of the apoplectic period; but this is not the rule, at least for the latter. Probably it is most common to have at first the skin reflexes abolished, while tendon and muscle jerks are increased. The front tap, ankle clonus, and toe jerk are often elicited. In fatal cases the skin, tendon, and muscle phenomena may be persistently absent. In cases which partially recover they are usually present, or return after having been absent. When consciousness

is not completely in abeyance, the special senses are often more or less blunted, especially on the paralyzed side. The uvula, velum, and tongue may or may not be deviated. The face presents great variations in appearance, being sometimes pale and drawn, but more often flushed. Vomiting is an occasional symptom in encephalic hemorrhage of all varieties, but it is most frequently present when the lesion is cerebellar. A true lobular or lobar pneumonia may develop during the attack and may complicate its symptomatology. Albumen may be found even when disease of the kidneys is absent, but when albumen and casts are both present, renal disease is practically certain. The urine may be of low specific gravity and sugar may be present. Jaksch states that, although transitory glycosuria has been reported after apoplexy, he was not able to find a case among fifty cases of fresh apoplexies within two years. Glycosuria is most likely to result when the fourth ventricle is involved in the lesion. Loeb believes that in most fatal cases of cerebral apoplexy sugar can be found in the urine, but that the excretion of sugar rarely continues longer than from twelve to twenty-four hours; also that albuminuria is frequently present, and always lasts longer than glycosuria.

Cortical Hemorrhage.—Subdural or intermeningeal hemorrhages are frequently also cortical, as they may invade the arachnopia and cortex, but a few cases may be more particularly classed as cortical or subpial. The hemorrhages are usually limited in size, and often take place in the arterioles or the capillaries. They may occur in connection with thrombosis of the longitudinal sinus, as in a case recorded by Horsley of cortical epilepsy with interesting spasmodic movements. These were turning of the head to the left, then raising the arm at right angles to the trunk in complete extension, with extreme extension of the wrists and interosseal position of the fingers, gradual turning of the head to the right, and subsequently the involvement of the rest of the body. In addition to thrombosis of sinus and veins, numerous cortical hemorrhages, varying in size, were found in the motor region of both hemispheres. Small, superficial, cortical extravasations of this kind are to be localized by the rules and principles of diagnosis for other irritative and destructive lesions of the brain surface.

Primary Ventricular Hemorrhage.—Ventricular hemorrhage may be primary or secondary. Primary ventricular hemorrhage is of comparatively rare occurrence. Its symptomatology differs according to its site and the severity of the effusion. When the hemorrhage is confined to one lateral ventricle, consciousness may not at first be lost. Monoplegia or hemiplegia may be present, but it is not invariably so, as is proved by a case reported by Dana. Commonly the blood passes into the lateral ventricles, and eventually into the third ventricle, iter, and fourth ventricle. The symptoms will at first be chiefly such phenomena as dazing or mental confu-

sion, dulness, and dizziness, but soon the symptoms of an extensive apoplexy will appear. Either hemiplegia or paralysis on both sides of the body may be present, commonly with rigidity and often with unilateral spasms or general convulsions. In a case of Dana's the patient was at first able to stand without help, both pupils were contracted and equal, neither paralysis nor anesthesia was present, and the patient could swallow and articulate; temperature was unchanged, and the knee jerks were normal. Two days later the temperature rose, and progressively appeared the symptoms of a profound apoplexy, such as unconsciousness, lapsing into deep coma, and stertorous and Cheyne-Stokes breathing. Both sides of the body seemed to be equally affected. At the autopsy blood was found filling the right lateral ventricle and extending into the third ventricle through the porta. The hemorrhage had taken place from a superficial vein in the outer anterior portion of the thalamus. Stupor and mental sluggishness were the chief characteristics of the symptomatology. Although ventricular hemorrhage is usually fatal, a few recoveries have been reported in cases in which the hemorrhage was small.

Secondary Ventricular Hemorrhage.—Secondary ventricular hemorrhage is the result of the breaking into the ventricles of blood which has been primarily extravasated into the solid substance of the brain. Its most usual forms are those which occur after rupture of the lenticulostriate, lenticular, or lenticulo-optic arteries. The extravasated blood forces its way into one lateral ventricle, and if the bleeding continues the whole ventricular system of the brain may be inundated. Occasionally the hemorrhage is from vessels of other regions, as in a case of my own in which primary hemorrhage occurred into the centrum ovale, the blood breaking through the roof of the lateral ventricle. The fourth ventricle is sometimes filled with blood in cerebellar hemorrhage. In the usual form of cerebroventricular hemorrhage, invasion of the lateral ventricle is announced by a deepening of the apoplectic symptoms. The unconsciousness becomes more profound, unilateral paralysis becomes absolute, and both sides of the body are soon involved; the patient no longer responds to sensory stimuli, tremulous and spasmodic movements are seen, and the breathing becomes of the typical Cheyne-Stokes variety. The phenomena are due to the displacement of the cerebrospinal liquid and the rending and stretching effects of both this liquid and the blood. Under the head of *ingravescent* or *progressive apoplexy*, Broadbent, Dana, and others have described cases which are in reality simply instances of intracerebral hemorrhage with secondary inundation of the ventricles. Some of the patients at first retain consciousness, and if they are kept absolutely at rest, and blood pressure is reduced, recovery is possible, by the stopping of the hemorrhage before the lateral ventricle is reached. A distinctive feature of these cases is the progressive deepening of the serious

phenomena. According to Charcot, next to the striatum, thalamus, and internal capsule, the most frequent seat of hemorrhage is the narrow band of gray substance in the external capsule called the claustrum, *avant-mur*, or teniform nucleus.

Hemorrhage into the Subcortex and Centrum Ovale.—Occasionally a small hemorrhage occurs immediately beneath the cortex, and this in the motor region may give special diagnostic features, as in one of my cases of hemorrhage with subsequent cystic formation just beneath the cortical areas for the leg and arm. The most persistent and distinctive symptom in this case was a continued spasticity of the paretic arm and leg. The right leg remained in an almost rigid condition, slightly flexed at the knee, and in a state very similar to that of both limbs in many cases of spasmodic tabes. The right arm was also markedly spastic, but the rigidity was not so extreme as in the leg. The fact emphasized by the record of this case is that a motor subcortical lesion partly destructive and partly irritative has for its most characteristic symptom a persisting spastic paresis or paralysis. Hemorrhage into the centrum ovale at points entirely removed from the cortex occurs with some frequency. I have observed cases of this kind in the prefrontal, frontoparietal, and occipital regions.

Cerebellar Hemorrhage.—A study of the points given under lesions of the cerebellum in the last chapter will help to make clear the symptomatology and diagnosis of cerebellar hemorrhage. The most usual sites are the vermis, one of the lateral lobes in the vicinity of the dentatum, and the medipeduncle. These hemorrhages may be difficult of diagnosis in the acute stage ; and cysts which remain after a small hemorrhage may give puzzling symptoms. Hemorrhages into any of the three localities above named may burst into the fourth ventricle and cause a speedy fatal issue. Added to early cerebellar symptoms such as vertigo, vomiting, astasia, or ataxia will be those of a severe ventricular apoplexy, the former being soon overshadowed by the latter. In one of my cases of cerebellar hemorrhage with ventricular effusion the blood passed from the metepicœle through the iter into the third and lateral ventricles, an unusual invasion. The patient was fifty-three years old, and the case had been diagnosticated as one of insular sclerosis. Two days before his death he began to feel dizzy and complained of headache. He was perfectly conscious, but he grew gradually worse, and in an hour was completely unconscious. His pulse was 72 and full. He had complete paralysis of the left side of his body, and on the chest on the left side fine muscular twitchings were observed. The pupils were slightly contracted and immobile. His temperature at the time of his seizure (one o'clock) was 96° F. ; at two o'clock it was 98.2° F. Before death he was cyanotic, and he evidently died of respiratory failure, the respirations ceasing a considerable time before the pulse.

No evidence of meningeal hemorrhage was present, but under the pia covering the posterior and inferior portion of the cerebellum was a slight extravasation of blood. The vessels at the base presented a few atheromatous patches. On opening the lateral ventricles a clot was found on the left side. The fourth ventricle was filled with black tarry blood. The main damage to the brain tissue was in the pons, crura, and cerebellum, which were ploughed up and disorganized. The heart was hypertrophied, but was without valvular lesions. The kidneys showed evidences of chronic parenchymatous nephritis. In several cases of what I believe to have been hemorrhagic cysts of the cerebellum I have seen the diagnosis of hysteria erroneously made. The histories were of apoplectic seizures brief in duration, with or without unconsciousness. One of these patients was left with an astasic and ataxic condition which involved both lower and upper extremities and to a less extent the trunk. She never regained the power of standing or walking without support. She also showed mental weakness and some ataxic disturbance of speech. In another case the symptoms closely simulated those of locomotor ataxia, but the patient had neither sensory nor pupillary changes, and her symptoms were clearly from a sudden seizure. Contrasting these two cases, in the first the knee jerks were exaggerated and the unsteadiness affected both the upper and the lower extremities ; in the second only the lower extremities were weak and unsteady and the knee jerks were abolished. The stigmata of hysteria were absent in both cases.

Multiple Encephalic Hemorrhage.—Multiple hemorrhages into the brain are not infrequent. They have been particularly observed in senile brains in which the vessels are more or less degenerated. Forms of focal hemorrhage may occur at successive periods, so that postmortem examinations may reveal a number of ochreous cysts in various parts of the brain and these evidently of different ages. In some autopsies symmetrical or nearly symmetrical hemorrhagic foci were found, which may have been either recent extravasations or the cysts left by former hemorrhages. It is comparatively common to find such symmetrical hemorrhages or the relics of them in the striata. Sometimes a focus of considerable size is found in one thalamus, and another of smaller size in the thalamus of the opposite side. Occasionally also such hemorrhages may be found in the centrum ovale in like or nearly similar positions on the two sides. Large and severe hemorrhages in rare instances take place simultaneously in the two hemispheres. In a case of Churton's, a large clot was found in the left thalamus and postcornu, and another of considerable size in the callosomarginal fissure invading the gyrus fornicatus and apparently producing complete anesthesia of the left foot. The pons is a not infrequent seat of multiple hemorrhage of small size. Recently a case of this

kind came under my observation at the Philadelphia Hospital. Three small hemorrhages and three old but minute cysts were found in one lateral half of the pons, and in the lenticula was also a cyst of considerable size. In many cases of injury of the head, either with or without fracture, I have noted hemorrhagic foci in various portions of the pia, and in less number in the substance of the brain. Similar minute hemorrhagic foci are sometimes found scattered throughout the brain substance and membranes in cases of very sudden and extensive hemorrhagic apoplexy. In one of my recorded cases of extensive capsuloganglionic hemorrhage with effusion of blood into the ventricles, extravasations and ecchymoses were present in other parts of the brain, very similar to those described by Duret, Park, and others in cases of traumatism, although not so numerous nor so extensive as in the latter, and not accompanied by so much contusion and laceration of brain substance. In this case a slight depression or splitting of the floor of the fourth ventricle, similar to that which has been observed in the traumatic cases, was also present. In such cases large masses of blood sometimes find their way to the central regions of the base, enveloping various cranial nerves and infiltrating the membranes and the spaces beneath them far out into the Sylvian, calcarine, and other fissures. Numerous punctate hemorrhages may also be found scattered through the substance of the brain. Conditions similar to those which result from external injury are brought about because the interior of the brain receives a violent blow when blood is suddenly poured out into its substance. Some regions seem to be peculiarly liable to extravasations, as the pia of the cerebellum and of the pons and oblongata and the pia and convolutions opposite to the seat of injury. The subject of multiple hemorrhage is of practical importance, especially when the hemorrhages occur simultaneously. The clinician must keep in mind the peculiarities of symptomatology which would result from such multiple lesions. When very decided symptoms of hemorrhage on one side of the brain are present, and in addition others which indicate pial or cortical irritation and some involvement of the other side, the complex phenomena are probably due to bilateral hemorrhages differing in size. In a case the recent symptomatology of which was that of thalamic and capsulothalamic hemorrhage, I found a hemorrhage in the right thalamus, a small cyst in the right striatum, and a large cyst in the left striatum. The vessels of the circle of Willis and its branches, especially the right medicerebral artery, were highly atheromatous, bluish white plates being seen on their inner coats. In the upper temporal region were a number of dilated vessels. I have found small hemorrhagic cysts in the striatum in cases which were under observation for several months, the patients never having presented hemiplegic symptoms.

Capillary Hemorrhage.—One of the subdivisions of encephalic hemorrhage is into *massive* and *minute*. Massive hemorrhages are those which take place from vessels of considerable size, and are such as have just been considered. Minute hemorrhages may be due to the rupture of arterioles or capillaries, or to transudation through veins. Hemorrhage from the capillaries is not usually classed as a distinct disease with a recognizable symptomatology, but it plays an important part in some cerebral affections. Capillary and arteriole extravasations into vital regions of the oblongata may be the cause of death in not a few chronic affections of the nervous system, as tabes, disseminated sclerosis, epilepsy, and in hydrophobia. Congested areas and small extravasations are frequently seen in the ventricular floor, especially in cases which die after convulsive seizures, or in which spasms have been frequently repeated during life. These foci often correspond to the cell nests of the cranial nerves, and especially to those from the fifth to the twelfth. In concussion of the brain, either with or without fracture, numerous capillary extravasations are found both in the brain substance and in its membranes. In leptomeningitis innumerable punctiform hemorrhages are often present, and these are supposed by some to be due to the influence of the specific virus of the disease. In the so-called red softening, whether encephalic or spinal, the red color is supposed to be due to capillary hemorrhages. When such hemorrhages are numerous in the same locality the condition is known as *capillary apoplexy*. A tendency to transudation or extravasation of blood from the capillaries and veins of the brain membranes is present in pernicious anemia, leucocythemia, and other blood affections. Nothing need be said in detail about the symptomatology, diagnosis, prognosis, and treatment of capillary hemorrhages. Their treatment will be that of the affection of which they form a part and in which they occur as episodes.

Duration and Complications.—The duration of a hemorrhagic apoplectic attack is variable. It depends upon the site, size, and extensions of the clot. A large hemorrhage into the pons and oblongata often results in immediate or nearly immediate death, constituting the so-called "thunderbolt apoplexy," *apoplexie foudroyante*. Secondary ventricular hemorrhage is usually fatal. In fatal cases death takes place most frequently on the second or the third day, but in cases in which apparently only one hemorrhage has occurred I have known death to result at any time within six or seven days after the seizure. In cases which last over three or four days bleeding usually recurs at the original site. As a rule, in fatal cases the patients do not recover consciousness and the course is consecutively from bad to worse, but occasionally there is partial recovery of consciousness with some improvement of the paralytic symptoms. When the patient recovers from the apoplectic attack, in periods varying

from one to several days, he usually begins to improve; the reflexes change for the better, motion and sensation—if the latter is disturbed—improve, consciousness is restored, and control over the evacuations is regained. Various symptoms clear up as the case progresses. In the majority of cases in about ten days or two weeks the patient simply shows the effect of the localized lesion, usually in some paralysis of motion or in loss of speech, and occasionally in some disturbance of sensation or of the special senses. A special section will be devoted in a later portion of this chapter to the hemiplegias, monoplegias, aphasias, and other chronic affections which follow apoplexies and other focal lesions of the brain. Edema of the lungs, or even a pneumonia of local or general type, may complicate an apoplectic attack. Sometimes the nervous symptoms simulate those of pneumonia and a mistake is made in this way, the shallow breathing being supposed to be due to lung complications, whereas it is in reality the result of the loss of cerebral control and is due to the hemorrhage

Etiology.—*Predisposing Causes.* The majority of cases of hemorrhagic apoplexy probably occur between the ages of thirty-five and fifty-five. It is, however, comparatively frequent in the first year of life, causing some of the cerebral palsies of children. Cerebral apoplexy occurs more frequently later in life in the rural districts than in cities, owing probably to the differences in modes of life. The curve of cerebral hemorrhage begins rather high, then drops and remains at almost a normal line until the age of fifteen, gradually rising until twenty-five, reaching its greatest height between forty-five and fifty-five, then sinking until the age of seventy-five is reached. (Dana.) Such a curve has only relative value and must be subject to many variations. Hemorrhage is more frequent in the male than in the female sex in the proportion of nearly two to one. It is not sex alone that leads to this disproportion, it is also the differences in life and occupation, which in men predispose to hemorrhage. Occupation has some influence. Berger and Schultze have shown that in some parts of Germany the workers in lead have an unusual predisposition to encephalic hemorrhage, as have those also whose occupation leads them to assume peculiar strained positions of the body. Sedentary, indoor occupation probably acts to a moderate degree as a predisposing cause. The influence of race and that of climate have not been well determined. According to some life insurance investigations, encephalic hemorrhage is, on the whole, most frequent among the Anglo-Saxon races. It is more common in winter than at other seasons. Among other predisposing causes of encephalic hemorrhage are an inherited tendency to arterial disease, acquired or hereditary syphilis, alcoholism, intemperance in eating with sedentary habits, life at high tension, and the introduction of toxic or infectious agents into the blood. It not infrequently

occurs in cases of renal, cardiac, or valvular disease, and it may follow the puerperium or infectious diseases. A tendency to arterial disease may be present in several generations of the same family. The underlying condition is in some cases a rheumatic, gouty, or other form of inflammatory and degenerative disease of the vessels. While acquired syphilis undoubtedly causes a fair percentage of cases of cerebral hemorrhage, it more frequently leads to thrombosis. Statistics and experience overwhelmingly demonstrate that chronic alcoholism is a strong predisponent to hemorrhagic apoplexy, although syphilis and alcoholism are so often found in the same cases that it is hard to discriminate as to their relative etiological influence.

Exciting Causes. It is well known that many cases of apoplexy occur as the immediate result of a debauch. A small percentage of attacks takes place during some especial bodily or mental stress, as from straining at stool, during coitus, and at times of great emotional excitement. In rare instances a hemorrhage into the brain follows a severe epileptic fit or a series of fits. Briefly stated, the most frequent exciting causes are those which increase blood pressure. The influences above stated are of this character, as are also others, such as attacks of vomiting, coughing, sneezing, straining exertions, and the lifting of great weights. Among other causes which may act differently but to the same end are a full meal, a cold bath, and sudden exposure to great cold or to abrupt variations in temperature. The apoplectic attacks which occur during sleep are more frequently due to thrombosis than to hemorrhage. If cerebral hemorrhage does occur during sleep, this may be due to the fact that gravity does not assist as much in the return of blood from the brain as from other parts of the body during this period.

Pathological Anatomy.—*Gross Appearances.* Much that relates to the pathological anatomy of encephalic hemorrhage has already been given in the discussion of the diseases which affect these intracranial vessels. Some opacity of the inner membranes of the brain not infrequently accompanies the widespread disease of the vessels. The amount of havoc wrought by the extravasated blood is to some extent dependent upon the condition of the brain tissues. Gray matter yields more readily than white, the tissues of the old, and those tissues which have undergone degeneration, more readily than those which are normal. The appearances of the clots and of the brain tissue which has been injured by them will therefore vary according to the size and the diffusion of the extravasation. One hemisphere may be distended and fluctuating, if the hemorrhage into its substance has been large. Sometimes the convolutions present a flattened and anemic appearance on the side of the hemorrhage. Small quantities of blood may be extruded into the Sylvian and other fissures from the interior of the brain. Autopsy in recent

cases of hemorrhage usually shows a large mass of blood destroying the middle and posterior portions of the internal capsule and adjoining ganglia, with a passage torn into the flooded ventricles; but occasionally, even in fatal cases, no blood is found in the ventricles or their annexes, although the structures which bound them are evidently distended on the side of the clot. Sometimes the appearances indicate a recent clot which has not quite broken into the ventricle, still having a thin roof formed by a layer of the caudatum or thalamus. The pia of the convexity may be edematous or opaque, and in spots and patches is often hyperemic. Usually in subpial and gyral hemorrhages the fissures near the seats of the ruptures will be found filled with blood, the convolutions being eroded but not greatly disintegrated. The blood is generally found in part beneath the arachnopia, which is often lifted and torn, and in part in the subdural space. It sometimes finds its way by gravity from the seat of hemorrhage to or nearly to the base.

Changes in the Clot. The blood, as indicated in the above records, may be fluid, coagulated, or partly coagulated. In coagulating, at first at least, the clot continues to fill the whole cavity which it has made for itself. Clotting takes place in the same way that blood coagulates in the heart, not as the process proceeds in a cup. (Charcot.) When death does not take place, granular degeneration of the clot soon begins, its various constituents gradually disappearing. The red corpuscles lose their coloring matter and remain only as hematoidin crystals. Leucocytes and fibrin also disappear. Step by step the clot condenses and separates itself from the fibrous lining membrane which lines the interior of the hemorrhagic cavity. The wall of the cavity undergoes changes, connective tissue increasing at the expense of degenerated nerve tissue. It gradually assumes a yellowish or ochreous color. The lining membrane may form as early as one or two weeks after the apoplexy. The clot dwindles more and more, becoming soft and yellowish, and after years it may disappear entirely. The cyst left may be of small or moderate or even of enormous size. In old hemiplegics with aphasia and contractures, I have seen cysts large enough to destroy the subcortex of the insula, the anterior two thirds of the capsules, the striate bodies, and a portion of the thalamus. These old and large cysts are usually filled with liquid and a small amount of detritus or of clot in the last stages of disintegration. They may be crossed by trabeculæ. When the hemorrhage has been very small, only a cicatrix may be left. Secondary degenerations frequently follow. They differ somewhat according to the site and extent of the hemorrhage, but are chiefly descending and of the fibres of the internal capsule and its continuation in the crus, pons, oblongata, and cord.

Diagnosis.—The diagnosis of encephalic hemorrhage must be considered with reference both to the apoplectic attack and to the

paralytic and other consequences of such attack ; but its more or less remote effects will receive special attention later. Before taking up diagnosis proper, it will be well briefly to call attention to the symptoms which threaten, and those which are supposed to threaten, an apoplectic stroke ; in other words, to describe what might be termed the prodromic diagnosis of encephalic apoplexy.

Threatening Symptoms. In the consideration of the clinical history, under the head of premonitory symptoms the phenomena given are sometimes recognized by the patient as the forerunners of an apoplectic or a paralytic attack ; but it is important to remember that these or at least similar symptoms may not be significant of such an event. They are to be regarded as forecasting acute hemorrhage or softening only when, with other symptoms such as have been detailed under diseases of the encephalic vessels (page 445), they are the evidences of arterial or arteriocapillary fibrosis. Ménière's disease, or forms of aural vertigo which are not in a strict sense this affection, cause symptoms, such as vertigo, tinnitus, and apprehension, which may call up the spectre of apoplexy ; but, as these disorders will be especially considered elsewhere, their diagnosis need not here be gone into at length. Mild neuritis or other rheumatic and gouty affections of the nerves and nerve centres may cause paresthesias or even pareses in some instances. The general good health of such patients, the history of a rheumatic or gouty diathesis, and the favorable response to treatment with the salicylates, lithium salts, or colchicum, will assist in making the diagnosis. Gouty disease of the vessels or membranes may after many years lead to apoplexy, but such symptoms cannot in a proper sense be regarded as the immediate prodromes of a cerebral attack. The lithemic and gouty, and sometimes the aged or neurasthenic who are not the victims of these diatheses, at times have spells of cardiac palpitation and irregularity with a sense of dissolution or danger, which they often believe to be the precursors of paralysis. The heart beat may be rapid or slow, and occasionally intermittent. Doubtless neither heart, vessels, nor nerves are in perfect condition, but such paroxysms may occur during many years without any more serious consequences than the weakness and dread produced by the attacks. Neurasthenics and patients in the early stages of melancholia often live in a fear of apoplexy, which is based on their uncomfortable sensations and general distress of mind ; but it is not necessary here to do more than to allude to this subject.

Affections to be differentiated from Encephalic Hemorrhage.—When the physician is confronted by an unconscious patient, and the diagnosis of hemorrhagic apoplexy is fairly presented, in the first place it must be distinguished from insensibility due to a variety of causes, such as alcoholism, uremia, diabetes, opium poisoning, saturnism, and other toxic conditions, sunstroke or heatstroke,

syncope, postepileptic stupor, hysterical coma, and pure simulation. Secondly, the different varieties of encephalic hemorrhage must be separated from each other, as intracerebral hemorrhage without ventricular effusion from ventricular hemorrhage, primary or secondary, intracerebral from meningeal hemorrhage, meningeal from cortical hemorrhage, and the different forms of cortical hemorrhage from each other and from large effusions. In the third place, encephalic hemorrhage must be differentiated from forms of apoplexy due to encephalic lesions, such as acute softening dependent upon embolism or thrombosis, intracranial abscess, or new growths.

Toxemias. In a case of uremic coma the patient may have swelling of the limbs, edema of the eyelids or of the face, the breath may have a urinous or beef tea odor, the pupils are generally dilated, and, as a rule not without exceptions, one side of the body shows more paralysis than the other. Considerable evidence has been accumulated to show that affections of the nervous system strictly limited to one half of the body occur during the course of some forms of Bright's disease. In this country Dercum has reported cases of hemichorea, hemiplegia, and unilateral convulsions. Raymond, Chantemesse, and Tenneson have reported a series of cases of unilateral affections, chiefly hemiplegia and epilepsy, apparently of uremic or at least of renal origin. In not one, according to the reporters, could a trace of a strictly focal lesion be discovered. Chauffard has reported a case under the title of uremic convulsions of the Jacksonian form. Such cases can be diagnosticated only by the history of the case and a full consideration of the signs and symptoms which indicate renal disease. Diabetic coma may be suspected if sugar is present in the urine, and especially if the patient has a history of glycosuria. Opium, chloral, lead, and other narcotic drugs or substances give rise to conditions of insensibility. Deep insensibility with contraction of both pupils, and the absence of indications of unilateral paralysis, such as conjugate deviation, loss of muscular tone, and absence of drooping of the face on one side, favor the diagnosis of opium poisoning; and yet Taylor has reported inequality of the pupils in one case of opium poisoning. Profound narcotism from opium and the coma of serious apoplexy present phenomena which are very similar. McEnroe, in speaking of the differential diagnosis between hemorrhage into the pons and opium poisoning, says that in pontile hemorrhage the coma is more profound. In narcotism it is possible to rouse the patient and to make him answer questions intelligently, as far as he will answer them at all. The pulse is full and strong in the early stages of opium poisoning, while in hemorrhage it is wiry, sometimes slow, sometimes rapid, but not usually full, strong, and regular. In the former the whole body is bathed in perspiration, and the respirations are less frequent than in hemorrhage. The discussion of the distinction between hemor-

rhagic apoplexy and the unconsciousness produced by chloral, cannabis indica, chloroform, hyoscyamus, or prussic acid and nitrobenzole, belongs to works on toxicology. A word might here be said about lead poisoning, as coma, convulsions, and other symptoms pointing to profound involvement of the brain are occasionally seen as the result of severe poisoning by lead; but here the presence of the lead line, the history of other forms of lead disease, and the occupation of the patient are of great value, if information with reference to these points can be obtained. Occasionally cases either of deep stupor or of excessive delirium are seen for which no cause can be assigned. These are sometimes due to toxemias of unknown origin. That they are not ordinary apoplexies can be recognized, but exactly what they are may be beyond the pale of diagnosis. In sunstroke, which by some is regarded as a toxemia, the fact that the patient has been exposed to excessive heat, the great rise and steady increase of body temperature, the prostrated or collapsed condition of the patient, and the absence of unilateral phenomena serve as differential features.

Alcoholism. A case of acute alcoholism may at the same time be one of apoplexy. One suffering from an apoplectic attack may have the smell of liquor on his breath and yet not be intoxicated. The breathing in a case of acute alcoholism, while it may be deep and heavy, is not truly stertorous nor of the Cheyne-Stokes variety, and examination will not reveal paralysis of one side of the body. Consciousness may appear to be lost, but this is not absolute, and the patient can generally be aroused, at least for a moment, from his stupor. The pupils are usually equal and dilated. The temperature may be two or three degrees below normal, but it does not show the successive variations of true apoplexy. Southey has recommended the injection into the rectum of a pint and a half of cold water with a tablespoonful of salt dissolved in it, which in his experience at once restored to consciousness a case of extreme drunkenness.

Miscellaneous Affections with Real or Apparent Unconsciousness. Asphyxia is to be distinguished by the presence of phenomena which show that the affection is primarily pulmonary and not cerebral. These are, in addition to the loss of consciousness, an embarrassed breathing, turgescence of the face, and frequently tremors and convulsions. Unilateral palsies are, as a rule, absent, as are also other phenomena which point to a lesion of one side of the brain. In fainting the face becomes pale, the patient loses consciousness, and usually sinks to the ground. The pulsations of the heart and the movements of breathing are diminished, and no indications of paralysis are present. A patient may be found in the deep stupor which commonly follows a severe epileptic paroxysm, and the physician have no knowledge of the preceding convulsion. Such a case may simulate closely hemorrhagic apoplexy, especially when an exhaustion paralysis of one side is present. If possible, a history should

be obtained before giving an opinion. The fact that a patient is in a profound sleep or stupor rather than in an apoplectic coma is usually evident; conjugate deviation of the head and eyes is commonly absent. That the tongue has been bitten points to an epileptic attack, which may also be indicated by the bruising of the body which has resulted from falls or the violent movements of the patient. In hysterical coma the breathing is not stertorous nor of the Cheyne-Stokes variety, and the face of the patient is usually placid. Hemiplegia or hemianesthesia, if present, is clearly different in character and depth from that of organic disease. Consciousness is perverted, but not profoundly lost. Inequality of the pupils and marked differences of the reflexes of the two sides are not present. Watchfulness, and the use of sensory tests and measures to throw the patient off guard, will generally suffice to make the diagnosis of simulation. Occasionally even the sleep disorders, such as somnambulism, trance, catalepsy, and the African sleeping disease, have been mistaken for apoplectic attacks with prolonged stupor. In such affections the clinical history, the protraction of the somnific state, the absence of paralysis, and the special phenomena of temperature, respiration, pulse, the state of the pupils, and ocular movements which are significant of apoplexy, will be of value. The apoplectiform attacks which occur during paralytic dementia are usually less severe and much more transient than ordinary attacks of intracerebral hemorrhage, but occasionally they are sufficiently serious to make the diagnosis of hemorrhage debatable. The previous history of such a case as to physical and mental symptoms should be carefully ascertained.

Differential Diagnosis of the Varieties of Encephalic Hemorrhage.—The diagnosis of different varieties of encephalic hemorrhage from each other has a value which is sometimes enhanced by the question of the advisability of surgical procedure. It is occasionally important, especially in cases which are known or suspected to be traumatic in origin, to differentiate between meningeal and ventricular hemorrhage. I have, for instance, known the diagnostic question chiefly discussed in an important case to be whether the patient was suffering from an intracerebral hemorrhage which had burst into the ventricles, or from an immense supradural or subdural clot. Under the symptomatology of primary and of secondary ventricular hemorrhage the chief diagnostic features of each have been fully given. In the secondary form the occurrence of the ventricular inundation is announced either abruptly or gradually by an increase and deepening of the phenomena of the attack. A distinct interval may occur between the first symptoms of an intracerebral apoplexy and those of a more pronounced character which announce the ventricular accident; in other cases the ventricular symptoms appear without any hiatus in the clinical phenomena.

Ventricular and Intracerebral Hemorrhage. The following table

from Grasset and Rauzier gives the main points of distinction between primary ventricular hemorrhage and intracerebral hemorrhage, the differential diagnosis of which, according to Gowers, is often practically impossible.

Ventricular Hemorrhage.	*Intracerebral Hemorrhage.*
Frequent in the old and the young.	More frequent towards middle age.
Onset rapid and violent.	Onset slower and less violent.
Generally very profound coma dating from the onset.	Coma generally less profound.
Convulsions frequent.	Convulsions rare.
Contractures frequent.	Contractures rare.
Paralysis often wanting; when present it may be general.	Paralysis the rule, and generally of the hemiplegic type.
Amelioration or a passing remission of the symptoms not rare.	Amelioration much less frequent.
Recovery a rare termination.	Recovery frequent.
Death rapid, often in a few hours.	Even in fatal cases life prolonged ordinarily for several days.

Ventricular and Meningeal Hemorrhage. Epidural or subdural hemorrhage, and in rarer cases cortical or even intracerebral hemorrhage, may occur as the result of severe injury, either as independent affections or coincidently with meningeal hemorrhage. The following table has been slightly modified from one given by Grasset and Rauzier, who included points determined by Ramskill and Nothnagel.

Ventricular Hemorrhage.	*Meningeal Hemorrhage.*
No antecedents to explain the lesion.	Antecedents: for the newborn a prolonged or hard labor; for the adult a traumatism.
Premonitory symptoms not rare.	Premonitory symptoms generally absent.
Gradually deepening unconsciousness.	When traumatic, unconsciousness followed by partial restoration of consciousness and then again by unconsciousness.
Cephalalgia rare.	Cephalalgia frequent.
Paralysis when present is usually hemiplegia.	Paralysis ordinarily generalized; hemiplegia exceptional.
Varying pupillary conditions.	Unilateral dilatation of pupil common.
Deviation of the mouth and tongue very common.	Deviation of the mouth and tongue rare.
Contractures very common.	Contractures, although frequent, less common.
Convulsions may be present, but not common.	Convulsions the rule.
Vomiting not frequent.	Vomiting frequent.
No symptoms of secondary meningitis with fever.	Symptoms of secondary meningitis with high fever towards the third or fourth day.
Death rapid.	Life, as a rule, prolonged several days.

To make the diagnosis from cerebral hemorrhage of an ordinary type, the history of an injury and its tangible evidences are of first importance. The methods of diagnosticating dural hemorrhages have already been given on page 270. It will be recalled that unilateral dilatation of the pupil on the side of injury to the head is an important sign in such hemorrhages; and so also is the occurrence of unconsciousness followed by a period of partial restoration of consciousness, and this again by unconsciousness. The elevation of temperature which is so important a diagnostic sign in distinguishing hemorrhage from acute softening may occur in almost any form of injury to the head, particularly if the brain substance is lacerated.

Sufficient diagnostic points have been given in the preceding pages in the discussion of cortical hemorrhage and of the different varieties of focal hemorrhage.

Differentiation of Encephalic Hemorrhage from other Acute Focal Lesions.—The distinction between hemorrhage into the brain and acute softening due either to embolism or to thrombosis may be important both for prognostic and for therapeutic reasons. After embolism and thrombosis have been considered, this diagnosis will be presented in tabular form. Reference will be made here only to a few points of difference. In favor of hemorrhage is the fact that an attack comes on between forty and sixty-five years; that the attack is not the sequence of endocarditis; that atheroma of the vessels is evidently present; that the seizure is sudden, and attended with loss of consciousness; and that certain peculiar changes in temperature result. Often the temperature shows an initial fall with subsequent more or less continuous elevation; often also the two axillæ and the two sides of the head show differences of temperature, it usually being higher on the paralyzed side. In acute softening the rise of temperature does not take place unless the lesion involves the pons or is very extensive. As against some of the points just given, it must be remembered that cerebral hemorrhage may occur at any age, that embolism may be sudden, and that disease of the heart and of the vessels is often present in embolic, thrombotic, and hemorrhagic cases. The method of making the diagnosis of encephalic hemorrhage from sinus thrombosis will be made clear by reference to the article on the latter (page 303). Here it is only necessary to call attention to the importance of a previous history of aural or other causative disease, and to the local signs, vascular and neural, of closure of particular sinuses. A brain tumor, so far as its symptoms are concerned, may remain largely latent, attention being first abruptly called to the affection by an attack of stupor or coma with or without paralysis. Frequently in such a case the ophthalmoscope reveals single or double optic neuritis. A history of headache or vertigo, or of other cephalic symptoms which did not

fix the attention of the patient, may be obtained on close inquiry. Blood is not infrequently extravasated into some forms of brain tumor, particularly gliomata, and occasionally such a hemorrhagic attack first reveals the presence of the tumor. Sudden paralytic attacks in tumor cases are not, however, usually due to the occurrence of such hemorrhages, but rather to the softening from pressure or the obliterative inflammation of the arteries which takes place around the tumor after it has progressed to some extent. In the differentiation of ordinary encephalic hemorrhage from a neoplasm, with intercurrent hemorrhage or with attacks of acute softening, the clinical history of the case and the presence of such distinctive symptoms as severe headache and optic neuritis will usually be sufficient to make the diagnosis clear. A patient suffering from syphilitic disease of the arteries, with gumma or gummatous meningitis, often has stuporous attacks which may be sudden in onset and may closely simulate the occurrence of an ordinary apoplexy. The diagnosis of brain syphilis of this type can be made only by a close study of the previous history of the patient and of his present and recent symptoms. The absence of the distinctive features—especially of the temperature, respiratory, and ocular phenomena—of an apoplectic attack will be helpful. An encephalic abscess will not often be confounded with hemorrhage. Occasionally after remaining for a long time more or less latent a large collection of pus may burst through its ordinary boundaries, causing an apoplectiform attack. The patient generally dies in a short time. In such a case careful investigation will reveal at least a few facts which point to abscess, as the history of purulent ear disease or of some suppurative process elsewhere, and irregular attacks of septic fever. In one of my cases an abscess was found occupying almost exactly the usual site of cerebral hemorrhage, and here the diagnosis was especially difficult; but this is a very uncommon site for cerebral abscess.

Prognosis.—When a patient is stricken with apoplexy, the two points about which information is usually asked of the physician by friends or relatives are whether the patient is likely to die in the attack, and what will be his probable mental and physical condition if the issue is not fatal. It is difficult to state in general terms the prospects of recovery in a case of cerebral hemorrhage, the prognosis depending upon the extent and location of the lesion, the previous health of the patient, and the care with which he is treated. Among conditions which point to a probable fatal issue are profound unconsciousness, paralytic involvement of both sides of the body, convulsions, great irregularity in the action of the heart, Cheyne-Stokes breathing, and marked initial fall of temperature with correspondingly great subsequent elevation. The formation of a trophic eschar on the buttock is of ominous import. Recovery is doubtful when the patient presents all or most of the above phe-

nomena, which usually indicate a large hemorrhage and one which has by extension become ventricular. When the disturbances of consciousness, respiration, pulse, and temperature are less marked, when paralysis is readily made out to be unilateral, and when rigidity and trophic eschars do not appear early, the chances of weathering the attack are better. As early recurrence of the bleeding takes place in a considerable percentage of cases, an absolute prognosis should, if possible, be withheld for several days. Incautious handling of the patient sometimes leads to fresh hemorrhage. The prognosis can occasionally be influenced by active treatment, particularly by treatment directed to the reduction of the blood pressure. The traumatic effect of a hemorrhage, often the most serious factor, is, according to Wernicke, equal to the product of the mass of the effused blood into the square of the rapidity with which it is poured out. The latter depends upon the pressure in the vessels, and can sometimes be partially controlled by appropriate treatment. Individuals respond very differently to the influence of cerebral traumatism, whether internal or external; in some, profound unconsciousness is produced by a much smaller clot than in others, and the prognosis in such cases will be uncertain. As to the exact amount of recovery in non-fatal cases, it is generally impossible during the first two weeks to give even an approximate opinion. Some patients pass through a severe apoplectic attack, recover consciousness and considerable vigor of mind and body, but remain absolutely bedridden for months or even years; others get so that they can hobble around with scarcely any further use of the paralyzed limbs than that which enables them wearily, and it may be painfully, to stand and walk; still others to a very large extent regain motor power, speech, and sensation, if the latter be affected. Mental integrity is very differently affected in different cases. Right hemiplegics are usually more emotional than those suffering from left sided paralysis. A large degree of mental power is often retained, but one who has suffered from an apoplectic seizure is never quite equal to his former self, and often is much below his normal standard in will power and in capacity for intellectual effort. A patient who will eventually walk again usually shows considerable improvement at the end of two, three, or four weeks. After a hemiplegic has recovered sufficiently to get about, it is difficult to say to what extent his improvement will progress. Vain and sometimes injurious efforts are made to compel further improvement, the patient often urgently demanding relief. The treatment, if judicious, may do some good, but the prognosis as to the amount of recovery is largely predetermined by the lesions. Apoplexy is regarded by Dana as a conservative agent, sometimes calling a halt to excessive activity and intemperate living and actually prolonging life; but it is doubtful whether this position is well taken, although it has some force so far as it bears on the change

in the personal habits of the patient due to the dread of another attack.

Treatment.—Whenever there is good reason to apprehend the occurrence of hemorrhagic apoplexy, the person threatened should lead a most careful life. In the majority of cases chronic degeneration of the arteries associated with cardiac and renal disease is present, and sometimes there may be syphilitic or gouty disease of the vessels; close attention should therefore be given to the kidneys, heart, and vessels. Food should be sufficient, but should be of a kind easily assimilated and digested. Constipation should be carefully avoided. Laxative mineral waters and lithia waters will be found of value. The patients should not lead lives that are too sedentary, and on the other hand violent exercise should be avoided. Alcohol should not be abused, and in most cases it is well to abstain entirely from its use. If hypertrophy of the heart or some other form of cardiac disease be present, a judicious use of cardiac remedies may be instrumental in preventing the apoplectic attacks. Arsenic and small doses of the iodides are of some value if long continued, because of their effects upon the vessels and viscera. High medical authorities hold that the best treatment in a case of encephalic hemorrhage is to let the patient alone, at least so far as either internal or external medication is concerned. Mendel believes that even the ice bag to the head is useless and that nothing should be done but to keep the patient at rest with the head somewhat elevated. Hughlings Jackson and others have advised only to administer one or two drops of croton oil. Position certainly counts for something in the treatment of such cases. As taught by Bowles, the turning of the patient to one side and keeping him in this position with some steady support will often prevent or modify the stertorous breathing, which is in part at least due to mechanical causes. A patient suffering from hemorrhagic apoplexy should be moved as little as possible, and when movement is absolutely necessary it should be effected with the greatest care, so as not to favor renewed bleeding. When the patient has involuntary evacuations from the bowels or the bladder, or when it is deemed necessary to use cathartics, he should be guarded as much as possible from violent exertion and unnecessary jarring on the part of those who handle him. It may be necessary to make use of urinals which can be attached to the patient. Dawbarn and Thompson recommend the application of the Spanish windlass to the extremities in cases of apoplexies, to lessen intravascular cerebral pressure and promote coagulation by preventing the return of venous blood to the trunk and head. Formerly the vast majority of cases of cerebral apoplexy were bled; now the reverse of this is true. Venesection may be called for in a few cases, when sthenic symptoms such as flushing or turgidity of the face with full, hard pulse are present.

In rare instances trephining has been tried for deep seated hemorrhages. Horsley has advised the tying of the common carotid artery for the ordinary type of cerebral hemorrhage, and Schwarz has recommended arteriotomy of the anterior branch of the temporal artery, on both sides if necessary, asserting that this will cause a reduction of the blood pressure in the carotids with less loss of blood than follows venesection.

ACUTE ENCEPHALIC SOFTENING.

Acute encephalic softening is due to death of the brain tissues, usually the result of occlusion of a vessel or vessels. The lumen of the vessel is obliterated from within by a plug or embolus carried from a distance, or by the gradual closing in of the vessel wall through proliferative disease and the formation of a thrombus. Embolism is from the Greek ἔμβολος, an embolus, derived from the verb meaning to throw or insert, and thrombosis is from the Greek θρόμβος, a clot of blood. Arteries may be compressed until their channels are obliterated by nodes, tumors, or exudates, necrosis of the tissue resulting. It probably also results from inflammation and manifold minute extravasations of blood; but, as a rule, the acute softening recognized as a special disease is dependent upon embolism or thrombosis. The popular idea of softening of the brain is largely erroneous. It seems to be believed that in some mysterious way general degeneration and softening of the brain result from strain, anxiety, and various causes which lead to serious nervous and mental disturbances but not to organic changes. Generalized softening practically never occurs, and nearly all softenings are due, as above stated, to the occlusion of vessels. Widely disseminated foci of softening may be present in a brain as the result of widespread arterial disease. All forms of acute softening are therefore local, but the affection may be *single*, *diffuse*, or *multiple*. Arterial obliteration does not always lead to softening. Numerous vessels of small size, or a few of considerable dimensions, may be occluded and softening not result. The parts are nourished by neighboring or by anastomosing vessels. The circulation to such parts is disturbed, and anemia and malnutrition may result, but necrosis may not take place or may be postponed until more extensive obliteration of the vessels occurs. The symptomatology of vessel obliteration is in part the symptomatology of arteriocapillary sclerosis or fibrosis, which has already been considered. In cases of thrombosis of large vessels the symptomatology of the focal softening which results is often complicated with that which is due to the diffuse disease of the vessels, many of the finer vessels being obliterated, although the vast majority are not. While obliteration of arteries is the most frequent pathological cause of softening, it is not the sole cause. The old writers undoubtedly made the mistake of supposing that it was almost

always due to inflammation, but the more recent schools of pathology have perhaps gone too far in the other direction. Summarizing, brain tissue may disintegrate and soften as the result of various processes: first by occlusion of arteries as the result of embolism or thrombosis, and occasionally from the pressure of growths or from compression in other ways; next comes the softening which accompanies encephalitis after it has reached a certain grade. Occasionally softening may be due to occlusion of veins, but such an origin is rare. Chronic softening results from other processes. Wernicke and Gowers have both given their allegiance to the view that we may have chronic progressive softening without obliteration of blood-vessels, and each cites cases in support of this doctrine. The centrum ovale both of the cerebrum and of the cerebellum was softened over large areas in these cases. It seems most probable, however, that these forms of softening are due to the obliteration of the fine terminal branches of the subcortical and ganglionic systems of vessels; but this can be demonstrated or negatived only by researches conducted with care, and by methods that will enable us to determine the condition of the most minute arteries.

SOFTENING FROM EMBOLISM.

Cerebral Arteries usually Attacked.—It is one of the best known facts in medicine that emboli more frequently lodge in arteries of the left than of the right side of the brain, giving therefore with similar frequency right sided paralytic affections. The explanation of this, at least so far as the mechanical conditions are concerned, is, that while the left carotid artery comes off nearly in a straight line with the blood current from the arch of the aorta, the right carotid branches from the innominate, which leaves the aorta almost at a right angle. Even in the parts of the brain supplied by the basilar and vertebral arteries, embolism is rather more likely to occur on the left side than on the right, because the left vertebral artery, which is larger than the right, arises from the highest point of the subclavian. The fact, however, that most of the blood passes by way of the basilar, which is a large azygous artery, before it is distributed from these arteries, makes the direction of the blood current and the peculiarities of the left vertebral supply less influential as regards intracranial lesions than are those of the left carotid. Embolism, thrombosis, and hemorrhage are all of more frequent occurrence in the left hemicerebrum. The left half of the brain, the leading half, pays for its supremacy by its greater liability to vascular disease.

Clinical History.—*Initial Symptoms.* Embolism is usually of sudden occurrence, and may be initiated with various phenomena. Occasionally, but not so often as in hemorrhage and thrombosis, it is preceded by vertiginous sensations. It is usually stated that pro-

dromata are absent, but this is not strictly true. Embolism often occurs in the absence of arterial disease, but chronic endarteritis and cardiac disease may be present, especially in cases in which a second or a third attack has occurred, and the prodromata are chiefly the same disturbances of circulation as precede thrombosis. Rarely the attack begins with spasms, usually local, and on the side which is later affected with paralysis. Loss of consciousness is frequent, its occurrence or nonoccurrence being related to the size of the vessels obstructed. In many cases the apoplectic attack is as sudden and severe as in cerebral hemorrhage, and is attended by as profound unconsciousness, but the coma, except in cases which have fatal issue, is commonly more transient than in hemorrhagic apoplexy. Vomiting is infrequent. As a rule, the evidences of vascular tension are not so marked as in hemorrhage. Delirium and fever may soon follow the attack, but these may be as much due to ulcerative endocarditis and to septicemic processes as to the direct effects of embolism. The initial fall of temperature so often observed in hemorrhage does not, as a rule, occur, and the rise of temperature which takes place is not so marked nor so distinctive in its features as it is in hemorrhage. Temperature observations are often valuable in the differential diagnosis of the different forms of apoplexy, but because of their complicating causes are not infrequently misleading. In thirty-eight cases of acute softening, Dana rarely found any disturbance of temperature on the first day, even in those which terminated fatally. On the second day a slight rise was often observed, so that the average was from 99.5° to 100° F. Dana says there are very few exceptions to the general law that the temperature rises after a serious hemorrhagic laceration of the brain and does not rise after a serious embolic or thrombotic softening of the brain. The rare exceptions to this clinical rule are when the embolic process is extremely large, involving an entire lobe, or when it is situated in the pons or the oblongata, or when it is due to a septic focus. In both embolic and thrombotic softening the temperature on the two sides is usually the same, or the variation is slight. Even during the apoplectic stage certain symptoms and signs may enable the physician to say that the patient is suffering from a focal lesion in a certain locality of the brain. Careful manipulation of the limbs of both sides may show less resistance or a decidedly flaccid paralysis in one arm and leg. The face may be seen by inspection to droop on one side and perhaps to be drawn towards the other. Conjugate deviation of the head and eyes may be present, but is not so frequent as in hemorrhage. Loss of sensation due to embolism of the ganglionic vessels, particularly when branches of the posterior cerebral are plugged, can sometimes be determined. The tendon and muscle phenomena are usually exaggerated on the side of the paralysis. As the patient rallies, consciousness is regained partially or altogether,

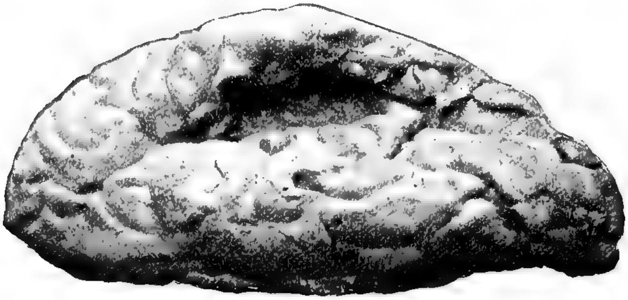
generally sooner than in hemorrhage. If the left side of the brain is attacked, aphasia is nearly always present.

Course. As regards both course and onset, encephalic embolism may be of several types. Most frequently, indeed in the majority of cases, the onset, as already stated, is sudden, and the acute stage short, at least as compared with hemorrhage. Some of these cases which show a large amount of paralysis at first largely recover from this paralysis, others remain permanently hemiplegic and it may be aphasic or hemianopsic. A few cases are rapidly fatal. Occasionally both embolic and thrombotic softening may occur without any symptoms having been recognized, or at least without symptoms which have been attributed to disease of the brain.

Focal Types of Encephalic Embolism.—Just as the most frequent description of cerebral hemorrhage is founded upon the symptomatology of hemorrhage from the lenticulostriate artery, so the usual account of an embolic apoplexy and of the acute state following it is founded upon the symptomatology of the results of occlusion of the same deep branches of the Sylvian or medicerebral artery. The acuteness of attack and the progress of a case of embolism must, however, vary with the locality affected; but to describe in detail each form would be simply to repeat much that has already been said in the pages devoted to cortical and interior localization. Among central vessels most frequently closed by emboli are then, first, the lenticulostriate, lenticular and other branches of the medicerebral artery; but closure of the lenticulo-optic artery, with softening involving the internal capsule, the postero-external portion of the lenticula, the anterior portion of the thalamus, and the tail of the caudatum, is also frequent. A gyral area which is a frequent seat of embolic softening is the left third frontal convolution, the insula, and the under surface of the overhanging portion of the operculum. Softening of the upper temporal convolutions is also comparatively common. In Fig. 278 is shown an immense cyst the result of embolic softening. It is not necessary to give a list of the different areas which may be isolated by processes of softening, as they correspond to the special vascular territories already considered. Occlusion of the lenticulostriate gives symptoms similar to those already given in the general description of the symptomatology of embolism; when the lenticulo-optic is affected, hemianesthesia and perhaps hemianopsia may be added to the motor symptom picture. When the vessels going to the insula and left third frontal are occluded (Fig. 279), aphasia will of course be a prominent result; when that supplying the first and the second temporal, word deafness may be the chief symptom. When large areas of the prefrontal lobe are the seat of embolic softening, peculiar psychical changes occur. Softening of the cuneus and of the optic radiations gives amblyopia or hemianopsia. Goldscheider relates a case of

embolism of the basilar artery, confirmed by autopsy, following ulcerative endocarditis, in which the main symptom was somnolence deepening into coma and ending fatally in twenty hours, the only other cerebral symptom being a contraction of the left pupil.

FIG. 278.

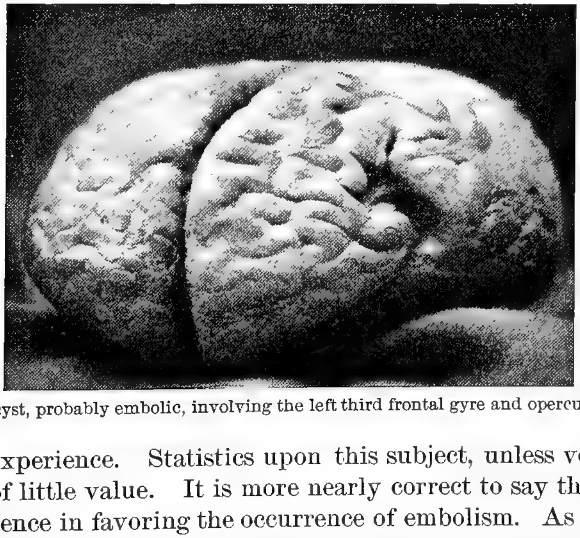


Cyst the result of embolic softening: the parts involved are the lower or Sylvian portions of the supramarginal and angular convolutions, the supertemporal convolution, and the insula.

As branches of the basilar supply the ventricular gray matter, the analogy of this case to acute bulbar polioencephalitis, in which somnolence is also a prominent symptom, is obvious.

Etiology.—Sex, age, and probably inherited peculiarities act as predisposing causes. Embolism is said to be of more frequent occurrence in females than in males; but this is not in accordance with

FIG. 279.



Old cyst, probably embolic, involving the left third frontal gyre and operculum.

my own experience. Statistics upon this subject, unless very extensive, are of little value. It is more nearly correct to say that sex has little influence in favoring the occurrence of embolism. As compared with hemorrhage and thrombosis, it is more often observed in the young, but it may occur at any age, dependent upon the disease which it accompanies or follows, as rheumatism, endocarditis, or sep-

ticemia. As these diseases, particularly the first two, are likely to originate in youth and middle age, so embolism is more common at these periods; it is of most frequent occurrence between puberty and fifty. Rheumatism, multiple neuritis, endocarditis, phlebitis occurring during the puerperium, and various other infectious diseases, such as diphtheria, scarlatina, and variola, act both as predisposing and as exciting causes. A relighting of old endocardial and arterial disease is a frequent exciting cause. A nervous shock may have, in exceptional cases, a tendency to the production of embolism. Thus, fright and violent exertion, particularly in the presence of recent endocarditis, may lead to the detachment of vegetations. A few very rare instances of echinococci plugging the cerebral vessels have been reported.

Pathogenesis.—The intruding embolus is usually carried from the heart, which has suffered from endocarditis and some form of mitral or aortic disease. It is derived more frequently from the vicinity of the aortic than from that of the mitral valves, but it is particularly likely to occur with mitral constriction. The slow flow of blood through the narrowed mitral orifice during diastole permits the aggregation of white corpuscles on the valve, and the quick flow during the auricular systole tends to detach the masses thus formed. Often there is great dilatation of the auricle, and a clot forms in the auricular appendix, fragments of which are likely to be detached, or the clot softens and particles from it pass into the blood current. The softened mass often contains micro-organisms. In ulcerative endocarditis the particles detached, which are usually small, may carry infective micrococci into the cerebral arteries. Minute vessels are therefore often plugged and the adjacent tissues necrosed. Other sources of emboli, besides a diseased heart, are aneurisms, particularly of the aorta and carotids, phlebitis following labor or of other origin, disease of the pulmonary veins, and ulcerative bronchitis and gangrene of the lungs. Minute pigment emboli sometimes block the cerebral capillaries, or fat embolism may follow degeneration of large vessels or disease of the bone. Certain poisonous substances, as carbon monoxide and phosphorus, are supposed to induce softening by their effect upon the vessels.

FIG. 280.

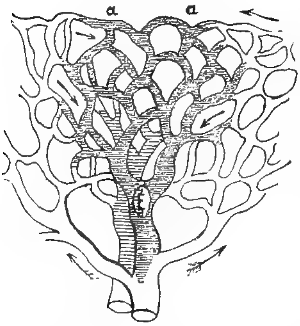


Diagram illustrative of the effects of embolic plugging: *aa*, portion deprived of its blood supply by the embolus E; A, artery; V, vein filled with blood clot. The arrows indicate the collateral channels which lead to a hyperemic zone around the occluded vessels. (Bevan Lewis, after Rindfleisch.)

Pathological Anatomy.—In embolic softening fat crystals and

granular débris are found on microscopical examination. In addition, leucocytes may be increased and the vessels may show slight inflammatory changes in their vicinity. The diagram, Fig. 280, illustrates the effect of embolic plugging on the neighboring vessels and tissues. Beyond the obstructed artery is the anemic wedge-shaped area of its distribution, and below the embolus are seen swollen branches, which tend to establish a collateral circulation. "If this fails, we get as a result engorgement of the latter vessels, and a congestive vascular zone surrounding the wedge-shaped area. The tissue here becomes swollen and œdematous, and minute hæmorrhages are apt to occur, whilst the whole central and peripheral texture becomes broken up by the effusion, and a true necrosis occurs of the tissue forming the area of distribution of the nutrient branch which has been plugged." (Bevan Lewis.)

Diagnosis.—The diagnosis of encephalic embolism, whether considered in respect to the apoplectic attack or with regard to the secondary chronic or paralytic stage, is often difficult, and sometimes must remain a matter of doubt, particularly as to its distinction from hemorrhage or thrombosis. The diagnosis at the time of the apoplexy is important, because the proper treatment differs somewhat from that which should be used in either hemorrhage or thrombosis, and especially in the former. It is important to establish the pathological cause of a hemiplegia, a monoplegia, or an aphasia, so as to adopt the best methods of protecting the patient from future attacks of endocardial disease and embolism. Age needs to be considered in arriving at a decision. Comparative youth is in favor of embolism. Here it must never be forgotten that many cases of syphilitic thrombosis occur before middle life, so that it is not improbable that apoplectic attacks leading to hemiplegia and occurring before middle age are frequently thrombotic in character and due to syphilitic periarteritis or endarteritis. A syphilitic history should be carefully inquired for, although it is not infrequent to meet with a history of both rheumatism and syphilis in the same case, and here the physician may be compelled to choose as seems to him best. As between embolism and thrombosis, abruptness of onset is one of the most important diagnostic points. An attack sufficient to cause marked paralysis usually comes on with sudden unconsciousness. As between hemorrhage and embolism in serious attacks, the temperature, as shown under hemorrhage, is a matter of great importance. The tabular presentation of the differential diagnosis of cerebral embolism from thrombosis will be given later. Too much stress should not be laid upon valvular disease. In those numerous cases of chronic disease in which nephritis, endarteritis, cardiac hypertrophy, and other pathological conditions are present together, the valves of the heart may also sooner or later become implicated ; but a patient of this kind is just as likely to have paretic or paralytic attacks as

the result of endarteritis and thrombosis as from cardiac vegetations carried into the circulation. One of my hospital cases, a woman about sixty years old, had cardiac degeneration with both aortic and mitral disease, and widespread atheroma of the vessels. This patient had three attacks of right sided paresis or paralysis, dying after the third one. Postmortem examination showed extensive calcification of the aortic crescents and degeneration of the heart walls. The aorta and the cerebral vessels were atheromatous. Centres of softening were found in the motor zone, in the brain, and also in other regions. Several of the secondary and tertiary branches of the middle cerebral artery were occluded as the result of disease in their walls and the formation of thrombi. In favor of thrombosis would be advanced age, atheroma of the vessels, fatty degeneration of the heart, and a succession of slight paretic attacks. In favor of embolism are youth, absence of signs of atheroma, previous history of rheumatism, and a sudden and comparatively severe attack of paralysis.

Prognosis.—The prognosis of encephalic embolism needs to be considered with reference to the apoplectic attack, the probability of the recurrence of the affection, and the persistence of the paralysis, aphasia, or other consequences. The prognosis of an embolic attack is better than that of hemorrhage, presuming that vessels of nearly equal size are affected. Even when the attack is severe the patient is more likely to recover from it than from a serious encephalic hemorrhage. As cerebral embolism sometimes takes place during the acute or subacute stage of an endocarditis, the prognosis as to the fatal issue and as to more or less speedy recovery will depend upon the state of the heart. Cases of embolism which at first have the appearance of great seriousness may on the clearing up of the apoplectic attack largely recover from the paralytic, aphasic, and mental symptoms. A patient who during the acute stage was totally hemiplegic and aphasic may in less than three weeks have left only a slight paresis of the arm and of the orolingual muscles; I have seen many such cases. Moderately severe cases may sometimes recover almost entirely from the paralysis. Most authorities state that attacks of embolism are not likely to recur, and this is probably true of embolism as compared with thrombosis; and yet I have somewhat frequently seen two and three attacks in the same patient. A patient who has once had a rheumatic and endocardial attack may as the result have only slight permanent changes, but will always be liable to a recurrence of the original affection in a more violent form and therefore also to a new or recurring embolic seizure. Cases which recover completely or nearly completely from the paralysis usually do so within a few weeks. After the chronic stage has set in, the difference in prognosis of the persisting symptoms as between hemorrhage, embolism, and thrombosis is not great.

Treatment.—The prophylaxis of encephalic embolism is in the first place the prevention of rheumatism and other infectious diseases which lead to endocarditis and the formation of vegetations, and in the second place the careful treatment of the heart and regulation of the circulation when evidences of old or recent endocarditis and valvular disease are present. One who has already suffered from rheumatism and endocardial disease should not be unduly exposed to great variations in temperature or to any depressing conditions likely to renew the old troubles. The remedies to regulate the circulation will of course vary according to the valves affected and the other special conditions present. Strophanthus, digitalis, cactus, on the one hand, or aconite and veratrum, on the other, may be needed. The maintenance of the general health of the patient by the use of tonics, rest, and careful regimen is important. The treatment of the attack differs somewhat from that called for in hemorrhagic apoplexy; but in both the patient should be kept perfectly quiet. In serious cases in which the breathing is interfered with the patient may be turned to one side and supported in this position in order to relieve the stertor. At the moment that the embolic obstruction occurs, all patent arteries dilate to compensate for the obstructed vessel. As long as this process lasts, a corresponding quantity of blood is used for their dilatation and so is lost to the rest of the cerebral circulation. The circulatory derangement is slighter the greater the force of the heart and the remaining available arterial tension. The greater the pressure in the veins the lighter the coma. A therapeutic deduction is to increase the force of the heart and the venous pressure by the recumbent position. (Geigel.) Hot applications to the extremities and trunk sometimes act favorably. To equalize and support the circulation, ammonium carbonate, digitalis, strophanthus, and cactus, with strychnine hypodermatically or by the mouth, may do much good, and alcoholic stimulants, usually in small quantity, may be needed. Venesection should not be used. If the patient is constipated, a quickly acting cathartic can be administered. The inhalation of oxygen has been used to tide the patient over a period of threatened collapse.

SOFTENING FROM THROMBOSIS.

Definitions.—Occlusion of an artery by the process known as thrombosis may, like embolism, also lead to acute softening of the brain tissue. A *thrombus* is a clot or plug which forms at a certain place in a bloodvessel, either as the result of changes in the blood or in the walls of the vessel, or usually as the result of changes both in the blood and in the vessel walls. Thrombi may form in the heart, sinuses, and veins, as well as in the arteries, but it is only with arterial thrombosis that we are here concerned. Sinus thrombosis has already been considered. Absolute obliteration of an artery

may take place without the lodgement of a clot, and in a strict sense such cases may not be regarded as instances of thrombosis; nevertheless, diseases which cause obliteration of bloodvessels by proliferative processes, or by pressure, produce softening and disturbances of the circulation. The distinction between a thrombus and an embolus is that in the former the obstructing mass forms at the seat of the occlusion, whereas in the latter the plug is carried from a distance. The disintegration or the partial disintegration of a thrombus may lead to embolism in the circulation at points beyond the thrombus, a thrombus again organizing at the original seat of the trouble: so that both processes may be present in the same case, and one may be causative of the other.

Pathological Varieties.—The thrombi which are found in encephalic vessels are variously subdivided. An obstructing thrombus is one which completely closes the lumen of the vessel; it may be incomplete or lateral, only partially closing the vessel, the adherent material clinging to its walls but leaving a channel of greater or less extent. Thrombi may again, according to their structure, be stratified or nonstratified, the latter being composed in the main of various layers of fibrinous material.

Etiological Varieties.—The varieties of thrombosis differ according to etiology. The term *simple thrombosis* is sometimes applied to those forms of the disease in which a clot forms in the vessels chiefly as the result of undue coagulability or of other pathological transformation of the blood, caused by depleting and infectious diseases, such as anemia, scurvy, the puerperal state, cancer, and by acute infectious and nutritional diseases in general. When disease of the vessel is present, it is sometimes named from the peculiar or special type of the disease: thus, it may be spoken of as syphilitic, gouty, atheromatous, etc. Pressure thrombosis is sometimes described, pressure of a tumor or an exudate compressing the vessel and giving rise to disease of its walls.

Clinical History.—*Premonitory Symptoms.* Premonitory symptoms are more marked in thrombosis than in hemorrhage or embolism. The same prodromes may be present in both hemorrhage and thrombosis, for the reason that both are dependent upon similar disease of the arteries. As a result of widespread syphilitic or atheromatous disease, patients may suffer from dull headache, vertigo, apathy, mental weakness, dizziness, and various disturbing and distressing sensations within the cranium. Some of the phenomena set down as prodromes of an attack of thrombosis of a vessel of moderate or large size are themselves due to obliteration of vessels of smaller size in various portions of the brain and at various periods. Among these so-called prodromic phenomena are transient aphasias and pareses, cranial nerve paralyses, partial anesthesias, and various cortical and subcortical phenomena of the special senses.

In thrombosis from atheroma premonitory symptoms are frequent. They depend on disturbances of the circulation caused by disease of the vessels, and may be present at intervals for a few hours, for weeks, or for months before the onset. The most common of these prodromes are dull general headache, giddiness, tingling, numbness, slight weakness in one half of the body, sometimes limited to a single limb, and often, but not always, corresponding in seat to the subsequent paralysis. Less commonly the patient shows defective articulation or some mental change, failure of memory, or irritability due to the malnutrition of the brain that is produced by widespread arterial disease. In syphilitic cases, as other lesions besides those of the vessels are present, the premonitory symptoms may be the same as those just described, but in addition more severe and general cerebral symptoms may be present. Headache is the most frequent; it is often severe, usually general, and may be worse at night; it may exist for weeks or only for a day or two before the onset. Sometimes considerable mental dulness or a somnolent condition may last for weeks. (Gowers.)

Onset. Hemorrhage and embolism have, as a rule, an abrupt onset, but in occasional cases both may come on slowly. Thrombosis, on the other hand, is usually of gradual onset, but occasionally comes on abruptly. It is commonly of gradual onset, because in the majority of cases the vessel lumen is nearly obliterated before the lodgement of the clot and complete closure take place; but when the blood dyscrasia and the velocity of the blood current play the most important part, a large orifice may be occluded abruptly and give a sudden onset and a comparatively severe apoplexy. As in the majority of cases the closure of the vessel is slow and progressive, that is, spreading from the original site of the occlusion backward until various branches of the first vessel obstructed are also occluded, the changes in the surrounding tissues take place more slowly, and consequently the paralytic, aphasic, and other clinical phenomena may come on without unconsciousness and in a less dramatic manner. When large vessels are closed they are none the less serious and persistent. In thrombosis, while the onset may be gradual, the progress of the case when once fully established may be rapid, although both gradual onset and gradual march are more common. The initiating symptoms of an attack of thrombosis will vary, of course, with the size of the vessel closed. Making our comparison with hemorrhage and embolism, if the vessel occluded is of considerable size, like a large cortical branch of the Sylvian, or like the lenticulostriate or the lenticular, or like a cerebellar branch of the basilar, the symptoms will be definite but of slow development. Motor paralysis comes on slowly, first in the leg and then in the arm and face, or it may be in the face and arm almost simultaneously, and last in the leg. Aphasia may be coincident with the lost power in the face or in one or

both of the members. Loss of sensation, absent at first, may soon be present and profound, especially if the vessels which go to the posterior capsular region are affected. The sequence of phenomena may differ greatly both as to the parts affected and as to the period elapsing between the beginning and the closing of the process. It is largely because of this peculiar manner of onset that patients are more likely to attribute their trouble to peripheral disease in thrombosis, and to brain disease in embolism and hemorrhage. In proportion to the surprise to which their consciousness is subjected are the patients likely to be affected with foreboding. When smaller vessels are occluded, the onset may be latent or light in phenomena. The patient may be conscious of a slight numbness or weakness in a limb or in the face, of a fugacious scarcely appreciable aphasia, of a transient amnesia for names or faces, or of a transient hemianopsia or amblyopia. Dizziness or a sense of uncertainty, and weakness, or incoordination, is often present.

Symptoms during the First Hours or Days of the Attack. As encephalic thrombosis—even when a vessel of considerable size is the seat of the occlusion—does not, like embolism and hemorrhage, give very striking apoplectic symptoms during the period of what may be regarded as the acute attack, the patient has not a sudden stroke, but finds himself paralyzed, usually at first partially, and he may succeed in getting to bed by his own efforts, consciousness, as a rule, not being lost. The temperature may fall slightly soon after the onset, and a little later may begin to rise, but the striking temperature variations of hemorrhage are absent. The range of temperature is not so great, the oscillations are not so violent; differences in both axillary and surface temperature on both sides may, however, be present, and are striking. Slight changes in pulse and respiration take place in the majority of cases. When a large focus of softening results from the obliteration of a large ganglionic vessel, the patient may have more marked disturbances of temperature, pulse, and respiration. Two of the factors which interfere with the vital functions of the brain in hemorrhage are absent in thrombosis. These are the irritative, distending, and pressure effects of hemorrhage, and, when the blood has passed into the ventricles, its immediate effects upon the important nuclei in the floor of the fourth ventricle. When the vertebral or basilar or any of their larger branches are the seats of thrombosis, special symptoms may be present even during the acute stage of the process. A well reported case with autopsy will sometimes most clearly indicate the symptomatology of thrombosis of a special vessel. Peabody has recorded a case of thrombosis of the basilar artery due to arteriosclerosis, with softening of the pons, in a woman aged thirty years. She was comatose on admission. One week before, she had complained of numbness and tingling in the right arm, and some impairment of power was present. Examination showed rigidity and

partial loss of power in the right upper and lower extremities, motor and sensory paralysis on the right side of the face, and dilatation of the right pupil. The heart sounds were feeble, and the temperature was normal on admission, but after four days it rose to 100° F. and at death it was 106° F.

Focal Symptoms. It has just been shown that according to the size and to some extent according to the location of a thrombus are the initial and immediate symptoms, general and focal. The early and the persistent phenomena, of course, depend upon the area of the brain necrosed as the result of the thrombosis. These differ in no essential respects from the phenomena of encephalic hemorrhage or embolism, and therefore do not need special consideration, except perhaps that a few words might be said about occasional cases which present a definite but restricted symptomatology. These are cases in which, usually because of syphilitic disease, vessels are nearly obliterated by gradual additions to their walls, and by this process and the lodgement of a thrombus small areas of softening originate. If these areas happen to be so situated as to destroy important nerve nuclei, tracts, or cortical centres, special symptoms may result. Paralysis of the iritic reflex or of accommodative power may be the sole symptom for a considerable time; more rarely a patient may suffer from total or partial word deafness; or the symptoms may be referable to lesions of the root fibres of the cranial nerves in the pons; or there may be hemianopsia, or paraphasia, or a special type of amnesia, as a persistent symptom. In brief, such a case may show one or two of the various phenomena which are eventually present in multiple softening to be presently considered.

Duration, Course, and Complications.—The active phenomena of the period of attack are fewer in thrombosis than in hemorrhage or embolism, and unless the vessel closed is very large and important the patient rallies in a few days. He may, however, be left with so large a degree of paralysis that he will not be able to get on his feet for several weeks; and indeed it is best that he should not be allowed to do this too soon. The further course of acute thrombosis differs but little from that of hemorrhage and embolism. The victims of thrombosis are perhaps more likely to show general mental enfeeblement, because their brains are badly nourished by the diseased vessels. Partial recovery from paralysis, aphasia, etc., takes place slowly; complete recovery almost never results. The symptomatology of thrombosis may be complicated by symptoms due to the renal, splenic, cardiac, and other diseases and degenerations which are often present.

Etiology.—One predisposing cause of thrombosis is age, with its degenerative accompaniments, such as a weak fatty heart, and atheroma of the vessels. Any constitutional condition, infectious disease, or toxic agent—rheumatism, gout, syphilis, alcoholism, poisoning by

lead or other substances—may lead to changes in the bloodvessels. Syphilis is the most frequent of these causes. Its influence in the production of disease of the vessels is sufficiently considered elsewhere in this volume.

Pathological Anatomy.—The vessels most commonly affected are the internal carotid, medicerebrals, vertebrals, basilar, and postcerebrals, but thrombosis may occur in any of the vessels of the brain, large or small. In old age minute terminals not infrequently become occluded as the result of obliterating endarteritis, with or without the formation of very small thrombi. Widespread changes in the vessels, often macroscopic, are present, and these may be any of the forms of disease already described in discussing affections of the walls of the encephalic vessels. The arteritis or atheroma may advance until the vessel is no longer pervious, but usually a clot lodges after the lumen of the artery has been much reduced. After closure has taken place, a forward and backward extension of the occluding process often occurs, so as to implicate various branches and connections of the original vessel. When the blood is in a pathological state, as during or after childbirth or infectious diseases, coagulation occurs with more readiness than under ordinary circumstances. As in embolism, anemia is one of the first effects of thrombosis, and later a portion of the anemic area usually softens. The appearances presented by the softened areas and their environment are much the same as those observed in embolism. Either red, yellow, or white softening may be present. The areas deprived of their blood are at first anemic, but after the first day break down. Capillary extravasation is the chief cause of the red tint. As the blood degenerates and its pigment is transformed, the yellowish color appears. Red and yellow softening are more frequent in the gray matter. In white softening, which may be present from the first, and is commonly subcortical, minute hemorrhages have not been so numerous.

Diagnosis.—Points in the differential diagnosis of encephalic arterial thrombosis have already been given in the discussion of sinus thrombosis and of encephalic hemorrhage and embolism. Hemorrhage, embolism, and thrombosis can at times be differentiated from one another, although the making of this diagnosis is sometimes difficult and exceptionally is an impossible task. In the diagnosis of thrombosis reliance is often largely on the past history of the patient, although the prodromes, modes of onset, and phenomena of the attack are of some assistance. The last are much the same for embolism and thrombosis; but previous history, premonitory symptoms, and methods of onset differ somewhat in the three affections. So much has already been said in the consideration of symptomatology that it will only be further necessary to summarize the most important differences in the table which follows.

Hemorrhage.	Embolism.	Thrombosis.
Most frequent between the ages of forty and sixty-five years, but may occur in infancy or at any age.	Most common between puberty and middle age.	Most frequent in the aged, the prematurely senile, and the syphilitic.
Often a family tendency to arterial disease.	Hereditary tendency to arterial disease not common.	Hereditary tendency to arterial disease occasionally present.
History of syphilis and a combination of renal, cardiac, and arterial disease comparatively frequent.	History of rheumatism, infectious disease, and old or recent cardiac disease.	History of syphilis very frequent.
Occasional premonitory symptoms, such as vertigo and temporary amnesia.	Premonitory symptoms rare.	Premonitory symptoms, indicative of chronic arterial disease, frequent.
Onset usually sudden.	Onset almost always sudden.	Rarely a sudden stroke; attack completed in a few minutes or hours.
Complete unconsciousness, sometimes brief, but oftener prolonged, and gradually deepening; rarely the patient does not lose consciousness.	Unconsciousness commonly complete but not prolonged.	Unconsciousness often absent.
Initial spasm very infrequent, but may occur.	Sometimes ushered in with spasm or convulsive movements.	Initial spasm absent.
Conjugate deviation of the eyes and rotation of the head common.	Conjugate deviation less frequent than in hemorrhage.	Conjugate deviation, affections of the pupil, early rigidity, paralysis, breathing, temperature, and pulse the same as for embolism.
Pupils generally fixed and often unequal.	Affections of the pupils less frequent.	
Early rigidity frequent.	Early rigidity less frequent.	
Hemiplegia commonly total and profound.	Paralysis usually less complete and less profound.	
Stertorous and often Cheyne-Stokes breathing.	Breathing less profoundly affected.	
Usually an initial fall with subsequent elevation of temperature.	Temperature changes less marked; initial fall often absent.	
Pulse varying, but often tense, hard, and slow.	Pulse likely to be weak, arhythmic, and compressible.	

Prognosis.—The prognosis varies considerably according to the vessel affected. When a vessel of large size, as the medicerebral, or one going to a vital centre, as a branch of the vertebral, is occluded, the effect produced may be permanent or even fatal. As regards the immediate result to life, thrombosis is ordinarily less serious than either hemorrhage or embolism; the prognosis as to the recurrence of the attacks is, however, worse in thrombosis than in the other affections. The diffuse disease of the vessels so commonly present

in the subjects of thrombosis is a constant menace, and is often the cause of more or less mental disturbance and deterioration.

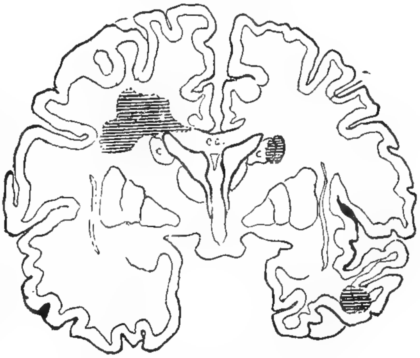
Treatment.—The preventive treatment of thrombosis is largely that of the diseases which lead to it, and is considered elsewhere under syphilis of the nervous system, diseases of the vessel walls, and the causal affections of organic diseases of the brain. When any of the forms of syphilitic arteritis and their visceral accompaniments are present or suspected, the patient should be treated with iodides, mercury, and ammonium salts. Arsenic is also beneficial, either alone or in combination with these drugs. Particular attention should be paid to the condition of the kidneys. An attack of cerebral thrombosis is rarely as threatening as one of hemorrhage or embolism. Often the patient does not take to his bed; commonly the attack is not attended with unconsciousness, the paralysis coming on gradually. The patient should be immediately put to bed. While this may not assist in removing the thrombus, it will do much towards tranquillizing the disturbed circulation. Probably the best treatment for the cerebral condition is that which has already been recommended under embolism. Efforts should be made to equalize and maintain the circulation, and this can be accomplished by the recumbent position, with the help of strophanthus, digitalis, and other drugs to which attention was called in the last section. Nitroglycerin, and even alcohol in moderate amounts, may be of great service. When the patient has rallied from the immediate effects of the attack, which is usually in a very short time, he should not be allowed to get out of bed. The bowels should be carefully regulated. Care should be taken in the use of cathartics in the treatment of thrombosis, for fear that purgation may weaken the heart and cause a tendency to increase in the size of the clot.

MULTIPLE ENCEPHALIC SOFTENING.

A few or many foci of softening may be found in the same brain. Charcot directed attention to the fact that in the badly irrigated territory in the interior of the cerebrum, where the terminal vessels of the cortical and central systems approach each other but do not anastomose, little lacunæ or lakelets of softening are likely to occur, especially in the aged. They are due to the obliterative atheroma or endarteritis which occurs in the old and broken down, and they are sometimes so small as to be scarcely detectable by the naked eye. Larger patches of softening may be found scattered through the brain and may have given rise to a series of attacks and an irregularly developing train of symptoms and conditions. The comparatively mild yet definite apoplectiform attacks often observed in the aged in the few years preceding death doubtless mark the occurrence of small foci of acute softening or hemorrhage, and other foci may originate without recognizable symptoms. It is not in the aged alone

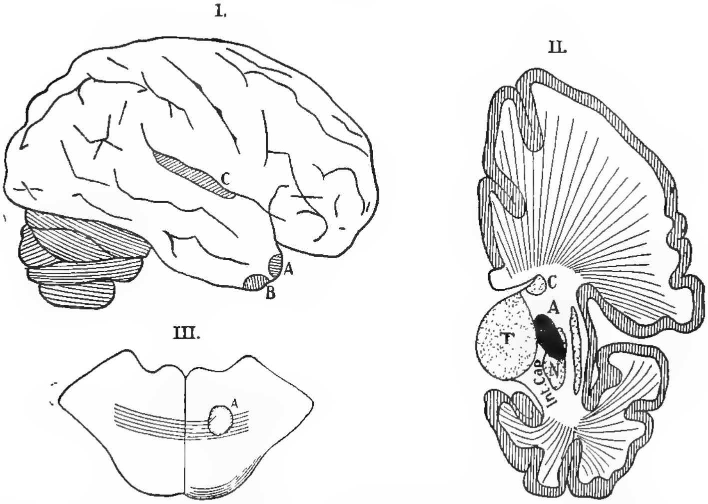
that multiple softening is observed. Cases associated with endarteritis at various ages have been recorded, as one by Herter, in a woman thirty-two years of age, without cardiac disease. Patches of softening of considerable extent were found in the prefrontal lobe, callosum, caudatum, lenticula, right temporal lobe, and left occipital lobe. Many vessels were examined microscopically and without exception were found to be the seat of endarteritis. Three of the foci of softening are shown in the sketch Fig. 281. Kolisco reports that he has found, as the result of carbon monoxide poisoning, small symmetrical areas of softening in the striatum, internal capsule, and thalamus, at points in the distribution of the terminal arteries which go to these structures. In Fig. 282 are shown several

FIG. 281.



Multiple foci of softening, (Herter.)

FIG. 282.



Areas of old and recent embolic softening in the same case: I., small foci of recent softening (A, B, C) in the right temporal lobe; II., old embolic cyst (A) involving the lenticula (N) and the internal capsule; T, thalamus; III., old embolic cyst in the central region of the pons at A.

areas of old and recent embolic softening in a case occurring in the service of Dr. Sinkler and the writer at the Philadelphia Hospital and recorded by Dr. E. A. Shumway in Volume III. of the Philadelphia Hospital Reports.

Symptomatology of Multiple Cerebral Softening.—This must be of varied and irregular character, depending upon the sizes and

sites of the affected areas. In a majority of cases the foci are in the centrum ovale and ganglia, but they are sometimes cortical. Mental symptoms, such as amnesia, impairment of attention, emotionality, apathy, and difficulties in orientation, are generally present. These at first are usually of a mild character, increasing in severity as the areas increase in number. They indicate disruption of associating and commissural fibres,—of the paths of communication which tend to preserve the solidarity and integrity of the mind. Lesions disseminated in considerable number throughout the cerebrum may give rise to such mental phenomena, although they are probably more marked and more special in character when they destroy prefrontal areas. As in Herter's case, multiple softening may show itself by a series of apoplectiform attacks with or without prodromata and with or without unconsciousness. In one attack the patient may be rendered hemiplegic or monoplegic; in another, aphasic; in another, a partial hemianesthesia or sector hemianopsia may occur, and in still another, amnesia and other psychical phenomena.

OCCLUSION OF THE ENCEPHALIC CAPILLARIES.

Capillary occlusion requires brief notice. Its diagnosis is rarely possible. Such symptoms as diffuse headache, vertigo, nausea, and general tremors and weakness are sometimes due to multiple occlusion of capillaries; and these symptoms may be either acute or chronic, according as they are dependent upon multiple emboli or multiple thrombosis more gradually determined. Occasionally acute delirium is the result of capillary embolisms associated with embolisms of the minutest arteries. Decided mental decay comes on as the result of arteriole and capillary occlusions. Among the forms of capillary and arterial embolism to which some attention has been directed are pigmentary embolism observed in the course of malaria; fat embolisms, the emboli having been derived from atheromatous vessels or from diseased or injured bones; and embolisms from pus cells or white blood corpuscles.

ENCEPHALIC INFARCTION.

The term *infarction*, derived from the Latin *infarcire*, to stuff, cram, or fill in, was first applied by Laennec to obstructed areas or infarcts seen in the lungs. Hemorrhagic and anemic infarcts are found in the brain, and have much the same appearance as infarcts in the spleen, kidneys, and lungs. They are found in all organs which have terminal vessels, in which they are commonly seen as wedge-shaped masses of necrosed tissue in and around which is more or less hemorrhagic infiltration. Pathological infarcts are by some regarded as originating at times simply from the extravasation of blood without occlusion of vessels. Charcot suggested that the cortex and underlying white matter may be divided into a system of wedge-

shaped areas, the apices of which are turned towards the central regions of the brain, and the base towards the surface, an arrangement of vascular subterritories which can be demonstrated by injections and by anatomical and microscopical examinations. Many superficial softenings of the cerebrum assume a somewhat wedge-shaped appearance, and infarcts are usually dark red, partly softened areas of this shape. The red color is due to extravasations, and these probably result from efforts of neighboring vessels to reestablish the circulation in parts cut off from their ordinary blood supply. Encephalic infarcts may be the direct or indirect result of embolism, but embolism and softening may be present without the formation of infarcts. A large area of softening in one place may be coincident with several infarcts in other localities, the latter resulting from multiple emboli. When proximal vessels—as branches of the medicerebral shortly after it is given off from the internal carotid—are plugged, cortical and subcortical areas may soften without infarction, but infarcts occur when distal branches are plugged, or when branches which deeply penetrate the cerebral interior are the seat of emboli.

ENCEPHALIC TUMORS.

Under the designation encephalic or intracranial tumor are included all new growths within the skull, whether they originate in the brain substance, in its vessels or membranes, in the periosteum, or in the bone, or whether they arise as secondary or metastatic processes, the primary lesion being in some other organ. Extracranial growths occasionally penetrate the skull and become in part endocranial and encephalic. The cranial cavity is the most frequent site of tumors, a greater variety of new growths being found here than in any other portion of the body. Bollinger found one hundred cases of brain tumor in eight thousand four hundred and eighty-eight bodies dissected in the Munich Pathological Institute in fourteen years, being one in eighty-five. Hale White found one in fifty-nine. The subject of brain tumors has become of increased practical importance since the possibility of removing such growths by operation has been demonstrated. The following table, by Starr, shows the percentage of cases probably removable to be about seven per cent.

Percentage of Brain Tumors Removable.

Author.	No. of Cases.	Operable.
1. Mills and Lloyd	100	10
2. Hale White	100	10
3. Starr	300	16
4. Knapp	40	2
5. Gray	102	10
6. Seydel	100	3
7. Dana	29	5
8. Starr	300	21
9. Byrom Bramwell	50	3

The literature of brain tumors is very extensive. The Index Catalogue of the Library of the Surgeon General's Office, in 1886, contained no less than six hundred and thirty-two references to contributions on intracranial tumor, and the number of such contributions has greatly increased since that time. Among recent works some of the most valuable are a prize dissertation by Knapp, a treatise by Bramwell, and monographs by Putnam-Jacobi and Starr. Starr's chapter in the Text-Book of Nervous Diseases by American Authors, edited by Dercum, gives references which bring the literature of the subject well up to date.

Etiology.—Heredity plays at least an indirect part in the causation of brain tumors, as when a tuberculous, sarcomatous, or carcinomatous taint is present. The frequency of occurrence of different forms of tumor will be considered under pathological varieties. The habits and occupations of men make them more liable than women to suffer from intracranial tumors: alcoholism, syphilis, and traumatisms, for instance, are more frequently present in the former. About two thirds of all cases occur in males. A large majority of cases occur between twenty and fifty; probably about one fourth of the whole number before the age of twenty. Gliomata and tubercular growths are frequent in children. Gummata develop oftenest from early adult life up to the age of forty-five; while sarcomatous, carcinomatous, and the rarer forms of tumor are more frequent near middle life. The aged are nearly exempt from all forms. While the influence of traumatism as an exciting cause of intracranial growths has probably been overestimated by some, it cannot be questioned that it is an efficient causative agent. The local contusions and extravasations produced by severe blows or falls on the head, even in the absence of fracture, may be the foci from which tumors develop. Frequently in my experience an apparently direct relation has existed between a head injury and the origin of a neoplasm. Certain tumors, as the fibromata, osteomata, and angiomata, appear to be of most frequent occurrence as the direct result of traumatism.

Pathological Varieties.—Ordinarily, in the present work, etiology, pathology, and morbid anatomy are considered after symptomatology, but in the case of brain tumors it may be best, for special reasons, to discuss them first. The symptomatology, for example, differs somewhat according to the pathological variety, and therefore some advantage accrues from previous knowledge of the latter. Intracranial growths are subdivided by Knapp into (1) the infectious granulomata; (2) connective tissue tumors; (3) epithelial tumors; and (4) aneurisms. Aneurisms have been considered in a special section. Under the infectious granulomata are included tubercular growths, gummata, and actinomyces. Following the German authors and Knapp, tubercular aggregations and gummata will be considered as tumors, although good pathological reasons might be advanced

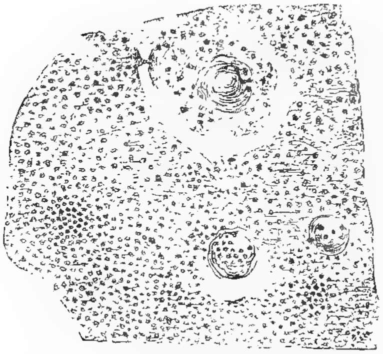
against placing them under neoplasms. By including them under infectious granulomata they are recognized as the results of inflammation which is dependent upon bacterial infection, and therefore they might be relegated to the class of inflammations; but the products of the inflammation are often so isolated and so aggregated as to constitute tumors in the clinical sense. They give rise to both focal and general symptoms, and need to have applied to them the same principles of diagnosis as sarcomata, gliomata, and some of the rarer neoplasms. It is best, therefore, in a work like the present to regard them as tumors, but it must be understood that in so doing we are not separating them from other inflammations. In the same sense that gummata and tubercular masses are classed as tumors, aneurisms and abscesses can also be so regarded, but, for special reasons, they are best discussed separately. In the class of connective tissue tumors are glioma, sarcoma, fibroma, osteoma, enchondroma, neuroma, and lipoma; while epithelial tumors are subdivided into carcinoma, adenoma, and cholesteatoma. In a table of one hundred selected cases prepared by Lloyd and the writer, gliomata, sarcomata, and tubercular tumors were represented by nearly the same percentages. According to Gowers, excluding syphilitic growths, tubercular tumors constitute more than one half the number of cases, and gliomata and sarcomata together about one third, gliomata being rather more frequent than sarcomata. Knapp, from an analysis of Birch-Hirschfeld's three hundred and fourteen cases, and of Starr's two hundred and ninety-nine cases occurring in children, and of forty personal cases, concludes that tubercle is the commonest form of intracranial growth; next the two connective tissue formations, sarcoma and glioma, with their varieties; and next, with a long interval, gumma, cancer, and parasitic cysts, other forms being exceptional. Experience teaches that the vast majority of new growths within the cranium originate either from the dura or from the arachnopia. Often the two membranes become so agglutinated that it is difficult to tell from which membrane the growth springs.

Tubercular Tumors. Tubercular tumors are often multiple, and may be present in widely separated regions of the brain, and in this way give rise to a puzzling symptomatology. As a rule, they are found in the membranes or along the cortical distributions of some of the larger arteries, as the Sylvian and postcerebrals, or their main branches. Occasionally they are observed in the ventricles or in the substance of the brain, probably here also originating from vessels, the bacilli being carried by the blood current to the infected spot. They are often irregular in shape, and may be constituted by a conglomeration of tubercular deposits. Their central portions often undergo cheesy metamorphosis, and the brain substance around them may be necrotic or sclerosed. On section they are commonly of a reddish hue in their more recently organized peripheral zones,

and yellowish or even greenish yellow in their older central portions. Under the microscope a few bacilli can sometimes be detected. It is sometimes difficult to distinguish a tubercular from a syphilitic growth, but the former, according to Bramwell, presents a marked difference from most syphilitic neoplasms, in that it is developed in the substance of the nervous tissue, while this is very rarely the case with the latter. Adjacent meningitis is frequently present.

Gummata. Most authorities hold that gummata do not develop as the result of inherited disease; certainly in the vast majority of cases, if not in all, the specific disease has been acquired. They differ much in size and appearance; thus, they may be nearly round, wedge-shaped, or of irregular contour. They also differ greatly in consistence, and Heubner has described two characteristic forms: the first a not sharply defined, comparatively soft, moist, grayish or grayish red mass, which on section exudes a scanty juice; the second a well demarcated, firm, almost cartilaginous mass, which is often dry and friable. On section of the latter the interior is often found cheesy and broken down, while the periphery is reddish and translucent, the appearances being strikingly like those presented by some tubercular growths. Connective tissue in large amounts may have developed in these growths. Vessels and nerve trunks in the vicinity of a gumma are not infrequently involved in the lesion. A gumma of the striatum with one of these forms of coincident involvement is shown in Fig. 283. Gummata are often multiple. They may develop at any time from a few months to a score of years after the primary infection. If recognized early they are amenable to active specific treatment. Most frequently the gumma is situated on the surface of the brain, originating from its inner or outer membranes, often having an adjacent zone of meningeal inflammation. Although growing from the membranes, it usually invades the brain itself, a process well represented in Fig. 284, from Bramwell. In Fig. 285 are shown the microscopical appearances of a gumma and its vessels, and also the infiltration of the surrounding tissues. In rare instances gummata develop in the substance of the brain, originating from the vessels. Bramwell has recorded two such cases.

FIG. 283.

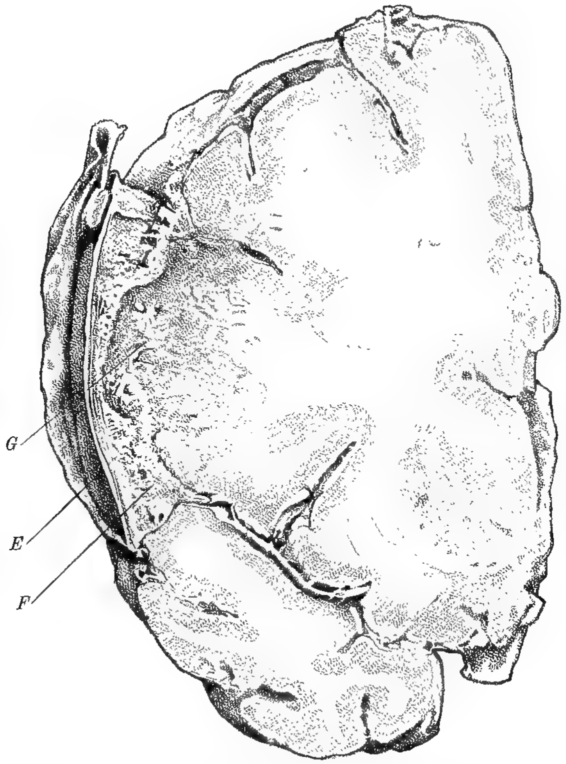


Gumma of the striatum, showing gummatous infiltration, and the hyaline metamorphosis of an artery with concentric infiltration of the cerebral tissue around the vessel. (Porter.)

Actinomyces. The parasitic fungoid organism called actinomyces in extremely rare cases directly or indirectly invades the brain.

Actinomycosis is the disease caused by this fungus. It often forms granulations which give rise to inflammations of the teeth, jaws, ears, neck, and face. Cases are reported of extension of the disease to the occipital foramen and into the brain, causing purulent inflammation and even abscess. Bollinger has reported an actinomycotic tumor as occurring in the third ventricle, the peculiar nodules of the specific microorganism having been found. In a case described by Delépine multiple tumors and abscesses in the cerebral hemispheres were caused by actinomyces.

Fig. 284.

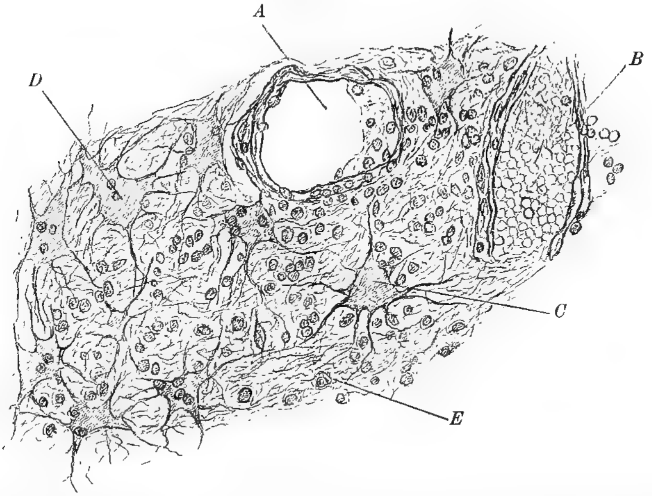


Transection through the left hemicerebrum in a case of gummatous syphilis: *E*, thickened and adherent dura; *F*, gummatous and inflammatory products between the membranes and the surface of the brain; *G*, surface of the brain at a point where it is being invaded by the syphilitic lesion. (Bramwell.)

Sarcomata. All varieties of sarcomata, as the round-celled, spindle-celled, giant-celled, alveolar, and melanotic, may occur in the brain. Commonly they originate from the bone, periosteum, or membranes, and gradually invade the cerebral substance. They are usually single, but may be multiple, in the latter case especially appearing as scattered melanotic nodules. Occasionally they are subcortical and infiltrating. Weir and Seguin have reported a case of this kind

in which a sarcomatous growth was found beneath the surface in the neighborhood of the facial centre. In the majority of cases sarco-

FIG. 285.



Microscopical section through a gumma of the brain: *C* and *D*, enlarged connective tissue cells; *E*, infiltration of the intervening tissue with corpuscular elements; *A*, transversely and *B*, longitudinally divided vessels. (Bramwell.)

mata not only are isolated but are also encapsulated. The macroscopic appearances, external and internal, and the microscopic structure, differ according to the variety. The spindle-shaped form is

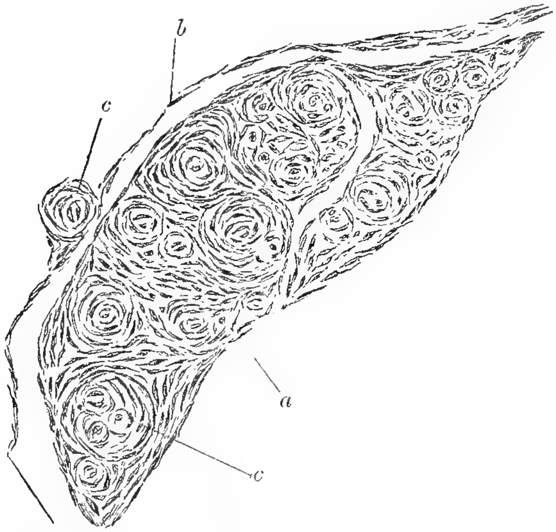
FIG. 286.

Large sarcoma of the left frontal lobe. (Morrison.)

generally firm and reddish; the melanotic is nodular and usually hard; while other varieties are soft, vascular, and sometimes gelatinous. In Fig. 286 is shown a sarcoma of large size, the case having been reported by Morrison. The growth proved to be a spindle-

shaped sarcoma, which was two and a half to three and one fourth inches in its greatest dimensions, and weighed five ounces after several months' immersion in alcohol. It was a dural tumor occupying the left anterior fossa of the skull, causing absorption of the brain substance as the growth advanced. It illustrates the great size attained by these neoplasms, the fact that they are usually encapsulated, and also one of their most common methods of invading the brain, by compressing its substance. At an early stage it might have been successfully removed. According to the relative preponderance of their constituent tissues the sarcomata found in the brain and elsewhere are classed as fibrosarcomata, gliosarcomata, myxosarcomata, and cystosarcomata. The *endothelioma* is a variety of sarcoma occasionally found in the brain. A tumor of this kind consisting largely of calcified cell nests, described by Hamilton, was as large as a walnut and

FIG. 287.

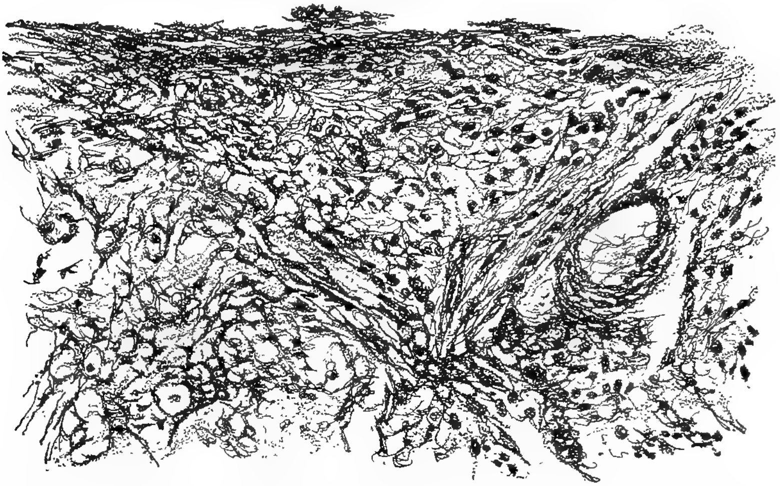


Microscopical section of an endothelioma of the left hemicerebrum: *a*, endothelial cells seen as small spindles between the cell nests, *c*, *c*; *b*, edge of a large mass of the tumor not shown in the figure. (McDowall and Bramwell.)

occupied the head of the striatum. In Fig. 287 is shown an unusually fine example of endothelioma from Bramwell's "Intracranial Tumors," the case having been recorded by McDowall. This tumor was situated at the under surface of the tip of the frontal lobe. *Myxomata*, or mucous tumors, are usually in part sarcomatous or gliomatous. The *myxosarcoma* is a comparatively rare form. Similarly *angiomata* are fundamentally either sarcomatous or gliomatous in nature. A view once held, but now not generally received, is that hemorrhagic pachymeningitis is a species of angiomatous neoplasm. The angioma is in reality an exceedingly vascular variety of sarcoma,

the *angiosarcoma*. Even the *psammoma*, or sand tumor, is in most cases a form of sarcoma, and has been called by Woodhead the *angiolithic* sarcoma. It has been found to contain spindle cells, but is largely composed of dark, gritty, calcareous material. Psammomata are most frequently found in the conarium, the choroid plexus, and the dura. Large calcareous plates are sometimes found in the latter membrane. In Fig. 288 are shown the microscopical appearances presented by the superficial part of a sarcoma removed by Prof.

FIG. 288.



Microscopic appearances of the superficial part of a sarcoma removed by Prof. Annandale from the motor area of the cerebrum. (Gibson.)

Annandale from a case under the care of Dr. Gibson of Edinburgh. This growth was situated in the motor region of the cerebrum, apparently growing from its inner membranes. It was of large size, and was mainly a soft sarcomatous mass, denser and older in the centre, presenting the usual characteristics of sarcomata found on the surface of the brain. It was interesting etiologically because it had apparently grown from a cicatrix the result of a traumatism received years before. The case is also interesting because the patient was greatly improved by the operation.

Gliomata. The gliomata occur with about the same frequency as sarcomata. They are of peculiar interest in the study of encephalic pathology, as they develop from the neuroglia of the brain substance, while the majority of encephalic neoplasms originate from the membranes, from vessels, or from glandular structures. They have been frequently observed in the cerebellum, pons, and oblongata, and in the cerebral hemispheres, and they occasionally develop in the retina. They originate oftener in the white than in the gray substance. The *fibroglioma*, *myxoglioma*, and *gliosarcoma* are common intracranial varieties. A glioma is often so ill defined from the brain substance

that on careless examination it is at first overlooked. Fatty degeneration may occur in the central (older) portions of the mass. The more vascular forms are peculiarly liable to intercurrent hemorrhage, giving rise to misleading apoplexies. Gliomata may be either soft or hard: the soft varieties are those which are most nearly allied to sarcomata. Osler found that gliomatous tumors studied by him contained very large spindle cells and coarse fibres. The hard varieties are more closely allied to the fibromata.

Fibromata. While fibromata are also of rare occurrence, one of the largest and most interesting intracranial growths recorded by the writer was of this character. The tumor had destroyed a large portion of the frontal lobe and other structures. Keen has also described a case of fibroma which was removed by him, the writer having been present at the operation. Fibromata are generally of a bluish or yellowish white color, of spheroidal shape and firm consistence. A vascular zone separates the tumor from the healthy tissue. They can be readily enucleated from the surrounding brain substance. Histologically they are composed of small fusiform cells, parallel to one another, with compact fibrillary intercellular tissue. They sometimes originate as the result of traumatisms.

Osteomata and Enchondromata. Osteomata, or true bony growths, are rare, and most commonly are projections from the inner table of the cranium; but the deposition of calcareous plates has been recorded as occurring in a variety of positions, as in the falx, tentorium, and other portions of the dura, and in very rare instances even in the substance of the brain. Enchondromata, or cartilaginous tumors, have been observed in the brain. They usually spring from the base of the cranium in the form of flattened masses arising either from the bones or from the dura and lying like plates upon the brain. They are composed of ordinary cartilage, and sometimes are in part bony, constituting the *osteo-enchondromata.*

Neuromata. True intracranial neuromata originate in the substance of the cranial nerves. False neuromata are fibrous growths springing from the nerve sheaths. The form of heterotopia described by Virchow as originating in the brain substance is not a neuroma, but a variety of glioma, sometimes designated as *neuroglioma ganglionare.*

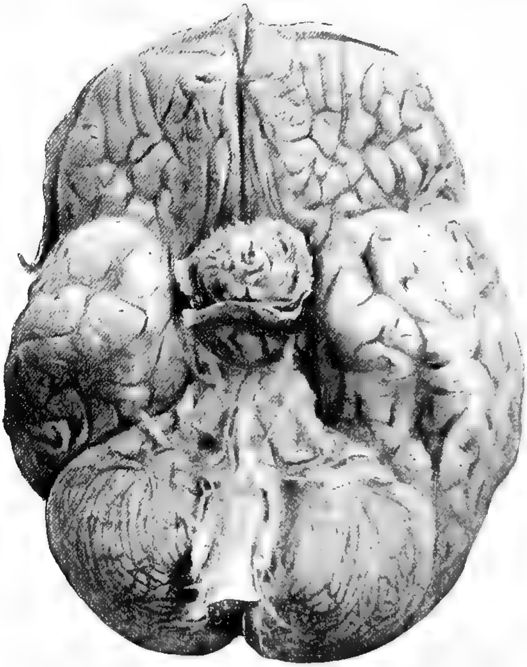
Lipomata. Lipomata, or fatty tumors, are rare within the cranium; but Taubner has recorded the occurrence of one the size of a hazelnut which grew from the quadrigeminal body. Bernhardt has also recorded a case.

Carcinomata. Carcinomata are epithelial neoplasms, and are not uncommon in the brain. They are often secondary. Bramwell has seen encephalic carcinoma follow cancer of the breast. Primary carcinoma has in rare instances been observed in the brain, usually having originated in the meninges or lining membrane of the ven-

tricles. Dural carcinoma is comparatively common. Carcinoma may be multiple, showing itself as scattered nodules in the brain or its membranes. Of its three varieties, the scirrhous, encephaloid, and colloid, the encephaloid is the most common encephalic form. Carcinomata are soft, elastic, fluctuating, and are not encapsulated. They are very vascular, and are made up largely of epithelial cells, presenting a stroma forming alveolar spaces in which are contained nests of epithelial cells, the so-called cancer nests. The evolution of an encephalic carcinoma may be very rapid. In a carcinoma of the base Wernicke observed a cancerous infiltration of the network of nerves situated in the neighborhood of the tumor.

Adenomata. The glandular tumors or adenomata in the brain are of course found either in the conarium or in the pituitary body (hypophysis). They are usually soft and spongy in structure. In one variety the tubules are lined with cylindrical epithelium, and another consists of acini lined with spheroidal epithelium containing a

FIG. 289.



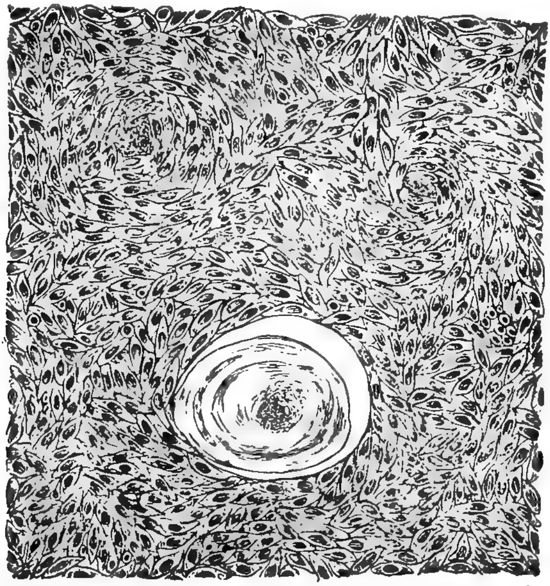
Adenomatous tumor of the pituitary body. (Blackburn.)

varying amount of connective tissue. Blackburn has described an interesting case of adenoma of the pituitary body (Fig. 289) which was nearly an inch and a quarter in its average diameter and almost globular in shape. It had exercised pressure upon the optic nerves, chiasm, cavernous sinus, and circle of Willis, had caused absorption of the posterior clinoid processes and other structures, and had pro-

duced a depression in the under surface of the brain. On section the growth was found to be soft, reddish gray, and moist, and proved to be chiefly an overgrowth of the epithelial portion of the hypophysis. The case was one of chronic dementia, the patient having been blind for some time; shortly before death she had a severe convulsion.

Cholesteatomata. Tumors of pearly lustre containing cholesterin, and called cholesteatomata, are found in various positions in the membranes of the brain, and more rarely in its substance. They are usually composed of proliferated endothelial cells of the arachnopia which have undergone a sort of fatty degeneration. They occur either solitary or as multiple nodules, and in some cases appear to be the remains of hydatid cysts. Dercum has recorded a case of unusual interest in which pearly bodies were found in the midst of a sarcoma. The microscopical appearance of a section of the tumor is shown in Fig. 290. The concentric onion-like structure of the cholesteatoma is readily seen.

Fig. 290.



Microscopical section of a tumor of the brain, showing a cholesteatoma in the midst of sarcomatous tissue. (Dercum.)

Tuberous Sclerosis. An interesting form of sclerotic pseudotumor is sometimes found in the brain, especially among the idiotic, imbecile, insane, and epileptic. It attains various sizes, from that of a pea to that of a walnut, or it may be as large as an entire convolution or a lobule. It is described by some authorities as tuberous sclerosis. The first examples of this affection seen by me were in brains obtained from the Pennsylvania Training School for Feeble-Minded Children. The sclerotic masses or nodules are sometimes

very distinctly defined, projecting from the brain like excrescences. They are due to an increase of glia.

Cysts and Cystic Degeneration.—Cysts found in the brain are usually porencephalic, the cysts of old hemorrhages, or small retention cysts in the vascular plexuses. Porencephaly will be considered later. In rare instances dermoid cysts have been found within the cranium. Kruse has reported an interesting case of this kind, the cyst not having caused any symptoms. The fourth ventricle was almost filled with a mass of hairs surrounded by a shiny, pasty substance. The tumor extended from the lower end of the ventricle to above the acoustic striæ. Kruse could find only two other cases in literature,—one in the cerebellum of a child of seven, with paraplegia, the other in the right striatum. A form of disseminated cystic degeneration of the brain is occasionally seen. Pick has recorded his observations on eight cases of this kind. Three were from cases of progressive paralysis, two from tabetics, one from a case of melancholia, and the other two showed no brain symptoms. The size of the cysts varied from that of a point barely visible up to that of a pea, and no lining membrane was detectable. Irregular compound cysts, formed by the coalescence of smaller ones, were found not only in the cortex but in the centrum ovale and ganglia. Most authors agree in referring these cysts to dilatations of the perivascular lymph channels. Occasionally a cystic tumor of large size is found in the ventricles, as in a case reported by Hirsch, in which the left ventricle contained a cystic tumor covering the thalamus, being eight centimetres long and six centimetres broad. The tumor, which was a typical spindle-celled sarcoma, had developed from the choroid plexus and had destroyed the posterior part of the caudatum and the posterolateral portions of the left thalamus.

Parasites of the Brain.—*Varieties.* Parasitic growths within the cranium include *cysticerci* and *hydatid cysts*, the most important class of the latter being the *echinococci*. These parasitic growths have been most observed in Germany, and seem from reported cases to be common also in Australia; in this country they are comparatively rare.

Cysticercus Cellulosæ. The term cysticercus cellulosæ is applied to the cystlike formations of the tenia solium in a certain stage of its development. These parasites enter the circulation from the intestines and are carried into the vessels of the brain. They vary greatly in size. The entire surface of the brain has been found thickly studded with them. Occasionally softening or inflammatory action takes place around them, particularly after the death of the parasite. By its series of hooklets the cysticercus attaches itself to the inner surface of a vessel, or to other tissues and organs. After having been located for a short time the hooklets fall off, and there is left a cyst or bunch of cysts. The symptomatology of cysticercus of the brain presents a few special features. The symptoms may

be of extremely varied character. The diagnosis will largely depend upon the history of the patient,—for instance, as to whether he is in the habit of eating uncooked meat, especially pork. In four cases recorded by Hirt all the patients suffered from epileptiform convulsions, and between the seizures they presented symptoms of mental confusion which were regarded as the psychical equivalents of the motor attack. All complained of headache and vertigo. A number of cases have been reported in which the parasite was found after death, but in which no symptoms were noticed during life, but the parasites were small, and they were located where active symptoms are not usually produced by lesions. Hydrocephalus has resulted from the presence of cysticerci which have been found floating free in the ventricles. Cases have been observed which in a general way presented the course and symptoms of paralytic dementia. Matignon has reported the case of a female found in the street aphasic and in collapse, who on admission to the hospital had generalized convulsions lasting from three quarters of an hour to an hour. The following day right hemiplegia with aphasia and relaxation of the anal sphincter was evident. In the third left frontal convolution a tenia cyst was found; on the anterior extremity of the left first frontal another; and on the inferior face of the cerebellum a third. The right temporal lobe contained a degenerated cyst the size of a walnut, with cartilaginous walls. In Fig. 291 is an illustration of a case of cysticercus cellulosæ at the base of the brain observed by Herdman. Early symptoms were paresis of the upper and, a little later, of the lower extremities. Knee jerks were exaggerated, and other motor, sensory, and ocular symptoms gradually developed. After a succession of chills followed by unconsciousness and a series of fits of short duration, the patient died. The autopsy showed a growth at the base of the brain in the middle region extending from the chiasm to the pons and spreading laterally on each side of the temporal lobes. The growth had the appearance of boiled sago, and was of pearly lustre, each individual portion being distinct. It seemed to originate in the meshes of the pia, and was adherent to the basilar artery and the arteries of the circle of Willis. Such a case might possibly be confounded with disseminated sclerosis of the pons, and this was one of the diagnoses made in this case, because

FIG. 291.

Cysticercus cellulosæ of the central region of the base of the brain. (Herdman.)

of the peculiar interferences with motion in the extremities, and the peculiar disturbances of speech.

Echinococcus. The echinococcus somewhat frequently invades the brain. It is usually found in an isolated mass, but may be multiple or diffuse. It is a hydatid, the *tenia echinococcus* of the dog, and is the most unevenly distributed of all the hydatids, being found in the liver and other organs of many animals. Its diagnosis is made from the history of the case. In some of the recorded cases many of the cysts in the brain penetrated to the outside. The most important symptoms of two cases were summarized in a collection of one hundred cases of brain tumor by Lloyd and the writer. In the first, recorded by Visconti, the parasitic cysts having been situated in the right occipital lobe, the symptoms were periodical headaches, pains in the scalp, neck, and vertebral column, staggering gait, diplopia, confused sounds in the right ear, and tetanoid contractions in the neck. In the second case, reported by Yates, an echinococcus cyst filled the whole of the left lateral ventricle, and the symptoms were periodical headaches, unsteady gait, left hemiparesis, peculiar swelling of the face, delirium, convulsions, and badly smelling discharge from the nose. Long and J. B. Hamilton have recorded a case of pedunculated polycystic hydatid found lying loose on the floor of the lateral ventricles but attached to the choroid plexus by a long pedicle. The patient was a right hemiplegic and aphasic, and the day before his death had what appeared to be an apoplectic fit ushered in by a severe convulsion. The effects of hydatids are chiefly those of irritation and pressure. In the brain after a time the hydatid dies and the sac sometimes dries up into a cheesy mass, which, however, can be identified by the hooklets, which may remain for a long time.

Common Sites of Certain Varieties of Brain Tumor.—It might be well to emphasize the fact that certain varieties of intracranial tumor are most likely to occur in special regions of the brain. When surgical procedure is contemplated the decision as to operation must depend, in part at least, upon the conclusion reached as to the nature of the growth. In my experience gliomatous tumors are most frequently cerebellar and occur especially in children. According to Bramwell, tumors of the pons are usually either scrofulous (tubercular), gliomatous, or syphilitic; tumors of the surface of the hemisphere, syphilitic, sarcomatous, or localized tuberculous deposits; and primary tumors of the centrum ovale and callosum, gliomata or sarcomata. "Tumors at the base are usually enlargements or new growths of the pituitary body, aneurisms of the large vessels, syphilitic gummata, cancers, or sarcomata."

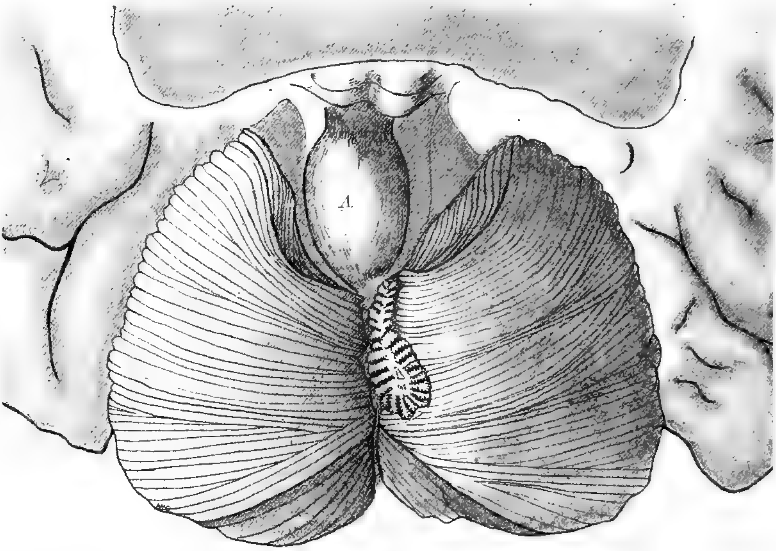
Clinical History.—*Cases without marked Symptoms.* The distinguished clinician and clinical teacher John Hughes Bennett was found after death to have a tumor the size of a hen's egg between the dura and the bone in the right temporal region. The growth had not

been suspected during life, although a depression in the skull was present two inches above the ear at a point over the tumor, and had frequently been noted. Other cases have been reported in which few or no symptoms were present, or were not discovered, during life. Doubtless in many of them unobtrusive phenomena were overlooked. Gliomata, and particularly the soft gliomata, when located in certain regions, such as the right temporal lobe, the prefrontal lobe, or a lateral lobe of the cerebellum, sometimes give rise to so few of the general symptoms of brain tumor that they pass altogether unrecognized, or are not suspected until very late in the progress of the disease. Tumors of rapid growth and firm consistence cause the most demonstrative symptoms. Idiosyncrasies may impress themselves upon the symptomatology. The psychical response of individuals to irritants differs greatly. Hysterical symptoms are so often pronounced as to mask the diagnosis and mislead hasty diagnosticians, a subject that will be further discussed under diagnosis.

Cases in which the Site or Extensions of the Tumor are not indicated by the Symptoms. In some cases the general symptoms of a tumor of the brain next to be described are present and marked, but symptoms which accurately indicate its site or at least its full extension are wanting. Occasionally such growths, as indicated in the last paragraph, may be located in the prefrontal, right temporal, or lateral cerebellar region, but they are often situated in regions which should give rise to distinct focal manifestations, yet these are wanting. While usually very close scrutiny of the symptomatology will reveal some symptoms which will help out the focal diagnosis, in rare instances such symptoms are entirely absent. Starr has recorded from his own experience, and has collected from neurological literature, some striking cases of this kind. One of his cases was a child with marked general symptoms of brain tumor in which the autopsy showed a large growth at the base in the median line involving the chiasm and optic nerves, resting upon all the motor nerves of the eyes, and compressing both fifth nerves. The child had no difficulty of vision and no paralysis of the cranial nerves. An infiltrating glioma recorded by the same author produced an apparent uniform increase in the size of the entire oblongata to double its ordinary dimensions, but showed absolutely no signs of any disease of either cranial nerves or tracts. Headaches, slight optic neuritis, occasional convulsions, and a slight staggering gait, led to the diagnosis of cerebellar tumor. A most interesting case in this connection was studied by Drs. Sinkler and Lloyd and the writer in the Philadelphia Hospital, and has been put on record by Dr. Sinkler in the third volume of the Philadelphia Hospital Reports. The patient was an Italian boy, fifteen years old, who had typical general symptoms of brain tumor, and at the time of admission to the hospital was blind from secondary atrophy after double optic neuritis. Among the few symp-

toms noted during his stay in the hospital were bulimia, a little uncertainty in gait, lessening of the knee jerks, slight retraction of the head, pain commonly referred to the occiput and neck, and pain above the clavicles and down the arms. The only cranial nerve symptoms were slight paresis of the right side of the face, sluggish reaction of the pupils, especially in the right eye, and a slight divergence in the left eye, this being a trifle higher than its fellow, according to Dr. Oliver, who made a careful ocular and ophthalmoscopic examination. The autopsy showed on the left side and dorsal aspect of the pons and oblongata a soft gliomatous mass, dorsal and ventral views of which are given in the illustrations (Figs. 292 and

FIG. 292.



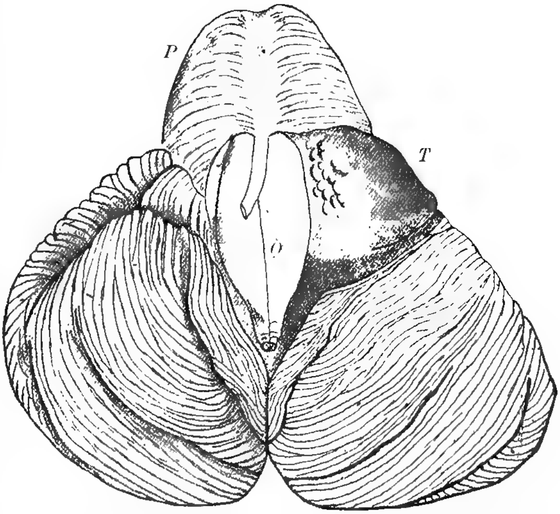
Dorsal view of a tumor involving the oblongata, pons, and cerebellar peduncles: A, outline of the tumor as it appeared when the fourth ventricle was exposed. The growth reached around the left side of the pons. The quadrigeminal body is seen irregularly diminished in size on the two sides.

293). This neoplasm involved the exits of the fifth, sixth, and seventh nerves, and to a considerable degree the regions of the nuclei and root fibres of these and other nerves. It extended so as to include the left prepeduncle (left anterior cerebellar peduncle). It was limited cephalad by the postgeminum. The mass was soft and brainlike in appearance and consistency, and, on opening it from above, extravasated blood was found in its tissue. It had infiltrated to a limited extent the tissue of the left cerebellar hemisphere.

General Symptoms.—Among the most important of the general symptoms of encephalic tumor—by which are meant symptoms common to growths of all kinds and in nearly all positions—are headache, vomiting, vertigo, insomnia, and double optic neuritis.

Mental changes, convulsions, temperature, and other febrile phenomena may be either general or focal symptoms according as they are due to lesions of special centres or to the influence of the neoplasms on the brain as a whole.

FIG. 293.



Ventral view of gliomatous tumor (same as Fig. 292) involving the pons, oblongata, and cerebellar peduncles: *T*, tumor mass; *O*, postoblongata, somewhat changed in shape from pressure; *P*, pons.

Headache. Headache varies much in character, but it is usually intense and often agonizing. Sometimes it is paroxysmal, but more frequently it is nearly continuous, with periods of great exacerbation. It is more constantly present than any other general symptom. Tenderness on percussion is of some service in fixing the site of a meningeal or cortical tumor, but even in these cases it may be misleading, and subjective localization of the pain is of little value for the purposes of focal diagnosis. Headache is somewhat less frequent in children. This is in part due to the fact that the growths in children are often gliomatous, and tumors of this class do not always cause headache. In very young children before the fontanelles have closed, headaches are not so likely to be present, or at least to be so violent, because, owing to the yielding of the cranial walls, the increase of tension is less. Syphilitic headaches are sometimes worse at night. In one hundred cases collected by Lloyd and the writer, headache was recorded in sixty-six, and in some of the others it was doubtless present but was not recorded. In only five cases was it stated not to have been present; and three of these were said to be gliomata. Putnam-Jacobi obtained nearly the same percentage by a tabulation of the cases of Ladame and Bernhardt. Headache was present in four hundred and one out of six hundred and fourteen cases. The chief causes of the pain are irritation of the nerves of the dura and stretching of this membrane.

Vomiting. Vomiting is present in a large percentage of cases. It may occur at any time and be unassociated with any disorder of the gastrointestinal tract. In cerebellar cases it is very commonly present, and sometimes of unusual severity. It is more paroxysmal,

occurring especially with exacerbation of the headache, and it may be brought on by movements of the head and body. Nausea without vomiting is occasionally present. In Putnam-Jacobi's tabulated cases vomiting was noticed in one hundred and sixty-two out of five hundred and seventy-eight cases.

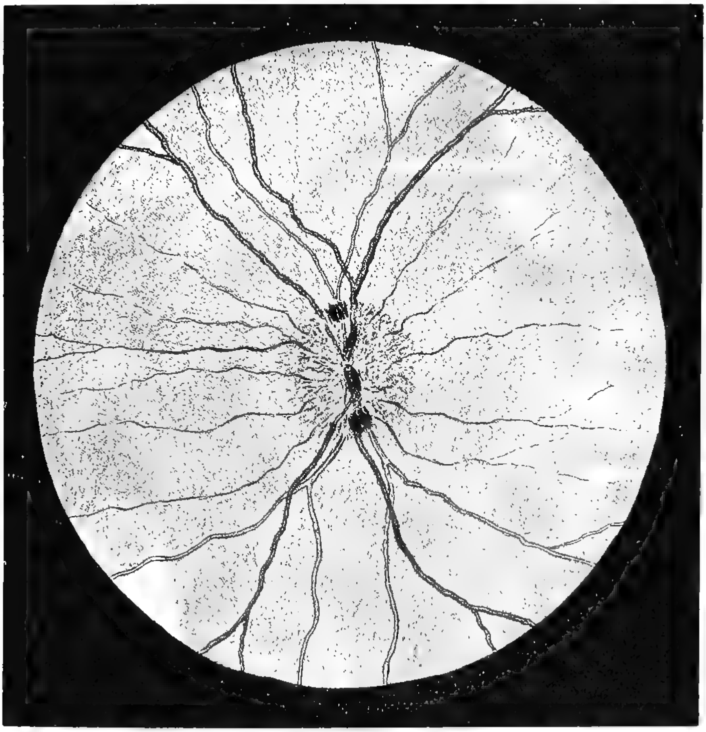
Vertigo. Vertigo is present in about one third of all cases, and, like headache and vomiting, is usually due to some irritation of the dura. In some instances it is ocular, in others it is due to irritation transmitted to the labyrinth from the cerebral cavities. Under the general designation of vertigo are included dizziness, giddiness, reeling, and feelings of insecurity not dependent upon true ataxia.

Insomnia. Insomnia, although often present, can hardly be regarded as one of the distinctive general symptoms of encephalic tumor. Like restlessness, emotionality, and irritability, it is often dependent upon the suffering caused by the disease. It is more frequently present in adults than in children, and is more likely to accompany certain forms of growths, as the syphilitic and the sarcomatous, than others. In rare instances the patient suffers from unusual somnolence instead of sleeplessness. This is especially true in intracranial syphilis, in which, in addition to the isolated growth or growths, diffuse lesions are present.

Papillitis. Optic neuritis or papillitis is a most important symptom of brain tumor. Following von Graefe, many writers on ophthalmology and neurology recognize two forms of optic nerve disease associated with intracranial tumors, one the so-called choked disk, the other a descending neuritis. The term neuroretinitis simply indicates that both nerve head and retina are implicated. As it is often impossible to separate the so-called varieties of neuritis from one another, it is probably better, as has been suggested, to include them all under the non-committal term papillitis. Of the various attempts to explain the production of this affection, the Leber-Deutschmann theory seems to have most in its favor. According to this view, papillitis is a true inflammation caused by the action of the irritant or infecting agent of the disease, which finds its way into the cerebrospinal fluid. In like manner it originates from the infection of a meningitis. Increased intracranial pressure serves to intensify and multiply the effects of this inflammation. Neuritis is probably present on both sides in from sixty to eighty per cent. of all the cases, but statistics on this subject are somewhat conflicting. Its presence is of great value in settling the diagnosis; its absence may prove nothing. Out of one hundred and seven cases of intracranial tumor tabulated by Edmunds and Lawford, sixty-eight showed optic neuritis. It is more frequent when the encephalic growth is in certain locations. More than one half of Edmunds and Lawford's cases were associated with tumors at the base or in the cerebellum. It has been noted in only about fifty per cent. in tumors of the motor cortex, and is rarely

present when the growths are confined to the hypophysis. Pituitary tumors do, however, sometimes cause neuritis and atrophy by their direct effects upon the chiasm and optic nerves. Single or monocular papillitis is rare, and when present is commonly due to a unilateral growth cephalad of the chiasm. Marked differences in the degree of neuritis present on each side are comparatively frequent. Vision is not always impaired at first, nor even for a long time, so that the ophthalmoscope should be resorted to when tumor is suspected, al-

FIG. 294.



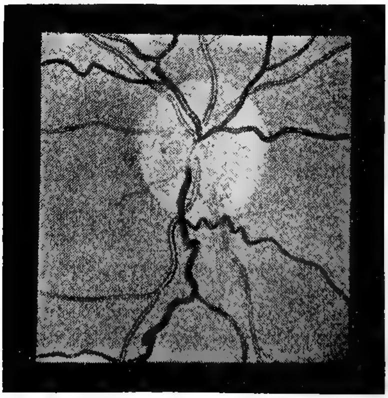
Fundus oculi (indirect image) in a case of well marked papillitis. (Bramwell.)

though the patient does not complain of his eyes. When blindness comes on with apparently great rapidity, papillitis may have existed unrecognized. Occasionally it is the only symptom of encephalic tumor discovered during the entire progress of the disease. A few cases of this kind with autopsies are on record, as one recorded by Morrow in which the patient had no symptoms except weakness, malaise, and occasional dizziness when walking, but the ophthalmoscope revealed double optic neuritis in the swollen stage with numerous retinal hemorrhages near the disk. A pear-shaped glioma was found in the right temporal lobe. In tumors of the prefrontal region papillitis may be the only positive symptom. Papillitis usu-

ally goes on from bad to worse, sometimes rapidly, but more often slowly. A partial recovery may take place, but the most common result, if the patient does not die while active neuritis is still present, is consecutive or postneuritic atrophy. Under diagnosis attention will be called to some of the difficulties met with in arriving at a decision as to the disease indicated, even when papillitis is present. Commonly, this is tumor of the brain, but it may be associated with other affections, such as leptomeningitis, encephalitis and abscess, and nephritis with albuminuric neuroretinitis.

Mental Disturbances and Deteriorations. Mental changes both general and focal are usually present in every case of encephalic tumor. Our concern here is with the former, although necessarily the psychic disorders caused by tumors in different localities will need to be referred to in order to make the subject clear. Among symptoms which rank as general are the mental disturbances due to the patient's suffering, and yet, as the pain may be caused by irritation of special branches of the fifth nerve, these symptoms are often indirectly local in origin. The patient is often highly emotional, restless, fretful, irascible, and easily affronted. After the disease has existed for some time he often becomes despondent, indifferent, and apathetic. Somnolence or semi-stupor, continuous or recurrent, is sometimes properly included among the general symptoms, but may be also due to the focal effects of large growths, especially when these are situated in the prefrontal lobes. Subcortical growths, which destroy cerebral association systems, often give rise to amnesia, defects in orientation, and inability to respond quickly to ideas or to peripheral stimuli. The symptoms thus induced are general in the sense that they interfere with the solidarity of brain action,—with the working of the brain as a whole. The brain is thrown into a more or less chaotic condition, causing disorderly psychic and other manifestations. Hallucinations, illusions, and delusions are most commonly due to the effects of tumors on special regions of the brain cortex or subcortex. Acute mania on the one hand, and profound depression or apathy on the other, is present in rare cases, and may obscure the diagnosis of brain tumor, especially if the patient is observed for a short period only. Hysterical and neurasthenic states are sometimes induced by tumors, and it may be by one the presence of which is not suspected until papillitis is discovered by

FIG. 295.



Regressive neuroretinitis of intracranial tumor. (Gowers.)

the ophthalmoscope. The diagnosis between hysteria and tumor of the brain, as will be presently shown, is not always easy. In children the mental changes vary with the age and intellectual development of the patients. Restlessness, fretfulness, irritability, apathy, and inability to concentrate attention and to pursue systematic study are frequently noted. In adults as well as in children, in studying the mental disturbances and deteriorations produced by a neoplasm, just as in the investigation of a case of insanity, the recent and previous mental status of the individual must be taken into account. He must be compared with his former normal self. Patients of little education and low intelligence, like many of those found in hospitals and infirmaries, do not show the fine differences and striking departures of a psychical character exhibited by the educated and intellectual.

Convulsions. General convulsions are common, and occur with special frequency in children. Although the convulsion is general, it often starts locally, but the local initiation of the spasm may be overlooked, or even when observed it may not be accorded its just significance as a localizing phenomenon. Such focally initiated general spasms are often the result of local irritation, as of some part of the cortical motor area, or of a sensory nerve in the dura. It is probable that the vast majority of cases have this mechanism; but it must be remembered that general convulsions, closely simulating ordinary grave epileptic attacks, may be due to tumors situated in any region of the brain. The irritation is radiated in all directions, eliciting or failing to elicit spasmodic phenomena, according to the resistance offered by the tracts which pass from the focus of disease to the motor cortex or bulb, and the stability or instability of these regions, stimulation of which gives rise to motor phenomena. Local spasms and the so-called Jacksonian epilepsies have received some consideration under cerebral localization, and will be again referred to when focal symptoms are discussed.

Pulse, Respiration, and Temperature. Aberrations of pulse, respiration, and temperature usually classed as febrile are common in encephalic tumors, although they may be absent or not attract attention except in the last stages of the disease. These febrile phenomena tend to come and go, and may even show some periodicity. The fever is doubtless due in some instances either to meningitis or to the toxemia which is an accompaniment of infectious neoplasms. The pulse shows great variations, these probably being conditioned by the size, character, and situation of the growth. It is often slow and arhythmic, especially in the early and middle stages of a slowly growing tumor. It not infrequently falls to less than fifty beats a minute. Towards the end of life it may become weak and rapid. When associated with meningitis, it may for a time be tense, hard, full, and frequent. Cerebellar, pontile, and oblongatal growths cause irregularities of the pulse through their more direct

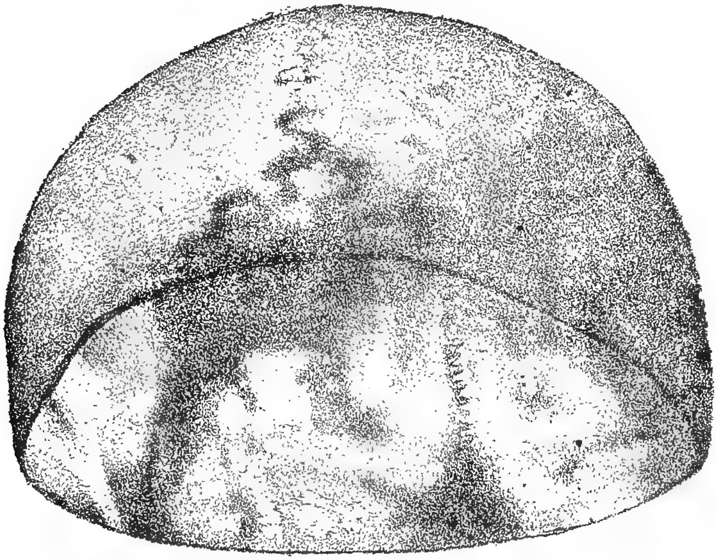
influence upon the centres of the vagi nerves. Now and then an extremely slow pulse is noted. Respiration, like pulse and temperature, may be very differently affected. In the terminal stages stertorous or Cheyne-Stokes breathing is common; the latter may be observed long before this stage is reached, occasionally as a recurring phenomenon, and especially with ventricular, basal, and cerebellar growths. As already stated, lesions of the tuber and its vicinity should give rise to rapid or to slow breathing, according as the lesion is destructive or irritative. Such respiratory phenomena belong rather to focal than to general manifestations. Unilateral differences in respiration are rare. Buccal, rectal, axillary, and surface temperatures all show departures from the normal during the progress of a case. If the entire course of the case except its terminal period is considered, the temperature is a little below the normal, but at irregular intervals the temperature shows marked elevations or vacillations. Sudden or rapid changes in temperature are sometimes due to increase of intracranial pressure, to aggravations of meningeal inflammation, or to an intercurrent extravasation of blood into the tumor. Differences in the temperature on the two sides of the body may be present and are occasionally quite marked. In one of my cases, a twin tumor in front of the chiasm, various interesting observations were made. The temperature was taken regularly twice daily in the right and left axillæ for eleven weeks before death, and gave an average for the right axilla in the morning of 99.1° F. and in the evening of 100° F.; for the left axilla 99.4° F. for the morning, and for the evening 101.4° F. On some days remarkable falls of temperature took place, as to 96° F., 95° F., and even to 94° F. and 93° F. The average head temperatures were above the normal; for stations on the right side of the head averaging about 97° F., and for the left side about 94.3° F. Elevation of the temperature of the head and scalp is occasionally present in large tumors. Lloyd and the writer concluded that in brain tumors the average temperature of the whole head is elevated several degrees above the normal, and that the elevation of temperature is generally greatest at the station nearest the seat of growth.

Changes in the Intracranial Percussion Note. In a few cases a procedure practised by Macewen may be of value in diagnosticating and locating a brain tumor, especially if of considerable size and situated at or near the surface. The method suggested is to tap the skull with the finger or percussion hammer and listen with the stethoscope placed upon the patient's forehead, pterion, or some other portion of his head. I have long practised percussion without the use of the stethoscope, holding my ear close to the head. Undoubtedly a peculiar note is present in many cases of hydrocephalus, the discovery of which may point to the presence of associated brain tumor. My experience does not lead me to rely with any certainty upon the

difference between the ordinary cranial note and that which is produced immediately over a new growth. The resonance is increased in hydrocephalus, and it may be found to be so decreased immediately over a meningeal or cortical tumor that the difference is recognizable. Sachs has noted this increased dulness in a few cases of encephalic tumor in children. Macewen regards the changed percussion note as also of value in the diagnosis of abscess.

Miscellaneous General Phenomena. Besides the general symptoms which have just been discussed in detail, others of more or less interest, and occurring with more or less frequency, may be present. Among these are constipation and other digestive disturbances, retraction of the abdomen, anemia, enlargement of the head, and thin-

Fig. 296.



Thinning of the calvarium in a case of cerebral tumor. (W. Hale White.)

ning of the bones of the skull. Constipation is very frequent, and is possibly due to the inhibitory influence of the tumor and the enforced inactivity of the patient. Other digestive disturbances may be present; the appetite may fail, or, on the other hand, it may be ravenous and a true bulimia may be present. As a rule, the patient emaciates. Children after a time become marasmic in appearance, wasting until they seem to be little more than skin and bones. The blood often shows on examination a true anemia. Great retraction of the abdomen may be present, as in a case reported by Hale White of gliomatous tumor in front of the chiasm in a child nine years old. This retraction is not due entirely to the loss of intra-abdominal fat, although this may account for it in part. It is a condition similar to

the scaphoid belly so often present in tubercular meningitis. In young children in whom the sutures and fontanelles are not fully closed, the head sometimes enlarges, owing to the increased intracranial pressure and consequent separation of the bones. The enlargement is due more to the hydrocephalus which accompanies the tumor than to any distention caused by the growth itself. In Hale White's case, although the patient was nine years old, the roof of each orbit was driven down a little, so that the upper eyelids became more vertical than normal, as is sometimes observed in hydrocephalus. The bones of the skull are occasionally thinned as the result of excessive and continued intracranial pressure. In Fig. 296 is shown a thin calvarium from a case of cerebral tumor reported by Hale White. The bones were transparent, and when held to the light showed their vessels as a beautiful arborescent network. The same writer refers to a specimen in the Museum of Guy's Hospital in which the thinning of the bone in a case of brain tumor actually laid open the tympanum. The thinning may in rare cases be so marked that the bones can be pressed in and will rebound like the "bottom of an oil can." Hyperesthesia is a comparatively common symptom. It is often present in the paralyzed limbs, and appears sometimes to be due to a secondary neuritis, and at other times to the central effects of the growth. The head also is frequently hyperesthetic.

Focal Symptoms.—*General Remarks.* The focal symptoms of an encephalic neoplasm—by which we mean those symptoms indicating destruction or irritation of a limited area of the brain with known functions which are capable of being interpreted by the effects of disease—may sometimes outweigh in importance, both for diagnostic and for prognostic purposes, the general symptoms, to the elucidation of which our attention has up to this point been directed. General symptoms, such as headache, papillitis, vertigo, vomiting, and convulsions, may tell us in no uncertain tones that a tumor is present, and yet may throw no light upon its location or its pathological character. The mere absence of focal symptoms is not sufficient to enable the diagnosis of the location to be reached by exclusion; for a growth located in any one of three or four extensive regions of the brain may give rise to no focal symptoms, or at least to none which attract attention. Such regions are the orbital surface of the prefrontal lobe, the right temporal lobe, or the lateral lobe of the cerebellum. Physiological and other studies in localization have been of practical service in establishing the focal symptomatology of brain tumors, and, on the other hand, much has been learned in regard to the functions of special regions of the brain through well studied and well reported tumor cases. Under cerebral localization, and in other sections of Chapter IV., the focal symptomatology of lesions of all kinds has been so fully presented that it will not be necessary again to consider the subject in much detail.

Cortical Tumors. Brain tumors may grow in or from any of the physiological subdivisions of the cortex given in the diagrams Figs. 228 and 229 (page 333), which represent the zones and centres of the lateral and mesal aspects of the cerebrum. These symptoms must therefore be considered according as they affect the prefrontal, the motor, the postparietal and limbic, the occipital, supertemporal, midtemporal, uncinate, and preorbital regions, or their known subdivisions. Careful study of the recorded tumors of the prefrontal lobe shows that they are usually accompanied by mental degradation or deterioration and special perversions of the higher psychical faculties. These cases need most careful study, particular attention being given to the previous history as regards intellectual capacity. When the growth is small it may be impossible to detect any focal symptoms, and when it occupies the orbital surface few distinct symptoms may be present, unless it extends caudad so as to involve the olfactory bulb and chiasm, in which case disorders of smell and sight may result. Bruns from a study of four cases of his own, and of cases of Oppenheim, Bernhardt, and others, concludes that disturbance of equilibrium, identical with the so-called cerebellar ataxia, occurs very frequently in tumors of the frontal lobes. This symptom he believes is much rarer in tumors of other parts of the brain and is absent with considerable regularity in tumors of the Rolandic region. The accompanying psychical symptoms and those due to pressure and irritation will usually enable a diagnosis to be made between cerebellar and frontal tumors when ataxia is present. Starr suggests that, as the prefrontal lobes are known to be connected with the cerebellum by special tracts, this symptom when present may be due to irritation conveyed by these tracts to the cerebellum. In my own experience this symptom has not often been present. In the central or Rolandic region the chief focal symptoms are monospasm or unilateral convulsions, with increasing paralysis of varying type according to the location and extension of the lesion. The spasmodic symptoms usually precede the paralysis in these cases. The spasm is often local, and generally begins in the same part in different attacks, as in the fingers, the toes, or the face on one side. Invasions of the hinder portion of the left third frontal and of the second frontal convolution give motor aphasia and motor agraphia which may be partial or complete according to the extent of the lesion. In the posteroparietal and limbic regions disorders of muscular and cutaneous sensibility are sometimes present, and can be looked for with certainty when the lesions are deep or when they are bilateral. When the gyrus fornicatus or the quadrate lobule is involved, in addition to anesthesia local vasomotor and trophic phenomena are also sometimes observed. (Savill.) Tumors of the anterior portion of the lateral occipital lobe, where it merges with the parietal lobe (angular region), give word blindness, and, if situated a little more posteriorly, apraxia, or soul

blindness. Lesions of the cuneus and of the calcarine region cause hemianopsia or quadrant or sector visual defects. When the tumor is situated in the superior temporal convolutions, partial or complete word deafness, and sometimes other affections of hearing, are present; while tumors of the third temporal convolution, as in one of my own cases, may give verbal amnesia with or without other symptoms (see pages 345–347). Tumors when located in the fifth temporal convolution and its vicinity cause disorders of smell, while affections of taste are supposed to be caused by tumors and other lesions of the fourth temporal convolution; but this point is not yet settled. The above summary is chiefly applicable, except for the motor region and the calcarine cortex, to lesions of the left hemisphere, and it must be understood that disturbances of psychical activity, motion, sensation, vision, audition, taste, and smell, include phenomena of irritation, destruction, and pressure. The phenomena of pressure and of irritation at a distance from a growth are sometimes spoken of as *indirect* symptoms.

Tumors of Special Regions below the Cortex. For the symptoms of tumors confined to the basal ganglia or invading special portions of the capsules and centrum ovale, the crura, conarium, hypophysis, quadrigeminal body, pons, and oblongata, it will simply be necessary to consult the preceding special sections on localization, and the sections and chapters which follow on diseases of the pons, oblongata, and cranial nerves. Tumors of the basal ganglia of the brain are seldom strictly localized to one or other of these bodies. Growths occurring in this region usually involve one or more of the ganglia and adjacent tracts, and can be localized only by a process of careful exclusion, as well as by the few special symptoms which they are known to give and which have already been discussed. With reference to tumors of such structures as the pineal and pituitary bodies, it is, as already stated, of great importance to bear in mind their effects upon neighboring parts. Symptoms referable to lesion of the chiasm, tuber cinereum, circle of Willis, and third ventricle are often present in pituitary tumors, while a growth involving the conarium may give symptoms showing invasion of the thalamus, midbrain, and other parts (see pages 367 and 368). The cerebellum is a frequent site of tumor. I do not believe it to be in any part a latent region, as is sometimes stated, and yet slowly growing gliomata, and probably some other forms of neoplasm, when they originate in the lateral lobes of the cerebellum may for a long time not present recognizable symptoms. Under "Functions and Lesions of the Cerebellum" (pages 373–382) the symptoms of tumor as of other cerebellar lesions are fully given; but one or two points might be re-emphasized. In tumors of this region some of the general symptoms of encephalic growths receive a special cerebellar stamp. The vomiting, vertigo, and optic neuritis, for example, are commonly of

an aggravated type, and symptoms referable to compression or invasion of the bulb or of the quadrigeminal body are frequently exhibited. The headache is often of occipital type, and nystagmus and other disorders of ocular movement are frequent. Tumors of the callosum, in accordance with what has been stated under cerebral localization, may give unilateral monoplegia, or hemiplegia developing into bilateral monoplegia or hemiplegia, spasms irregularly bilateral, hebetude, slowness of speech, and irregular gait. The bilaterality of the symptoms will sometimes assist in the diagnosis. Dementia is frequent, and the general symptoms of encephalic tumor are nearly always present.

Multiple Tumors.—The possibility of multiple tumors should never be forgotten. They have been reported as occurring in any number from two to a score. Tumors most frequently multiple are the tubercular, the syphilitic, and the sarcomatous. Occasionally neoplasms show a tendency to be symmetrically located on the two sides of the brain. Multiple tumors may, of course, give phenomena referable to various parts of the brain, symptoms and signs so conflicting as to make local diagnosis impossible, and even to confuse the expert as to general diagnosis. In other cases, however, one growth of large size or in an active region takes command of the situation, and leads clearly to its diagnosis in spite of other less significant tumors. In one of my cases not only was the presence of a large tumor of the motor cortex diagnosticated, but its exact location was indicated during life. Postmortem examination, however, showed a smaller tumor at the inferior angle of the right lobe of the cerebellum, and also some basal meningitis with effusion, which had not been suspected. When operation is in question, the possibility of the coexistence of another tumor should be carefully weighed. In one case three gummata were found by me,—one in the prefrontal region, another in the retrocentral fissure, and a third in the supramarginal convolution. The general symptoms of brain tumor were present, but no localization was possible. Occasionally all the ventricles are invaded by sarcomatous growths.

Duration.—No very positive statements can be made as to the duration and course of encephalic tumors. The duration may depend on various factors, such as the nature of the growth, the age and general health of the patient, and the care which can be given him. Gliomata and encapsulated sarcomata are often of long duration. (Bramwell.) In thirty-two cases of brain tumor studied by Knapp the duration of the shortest—measuring duration from the time of the first recognition of the symptoms—was twenty-five days, that of the longest twelve years; the average duration being sixteen months. A case of cerebellar tumor recorded by Seguin had definite symptoms for eighteen years. Andral has recorded a case which lasted fifteen years, and other cases lasting ten or twelve years have been put on record; but these are exceptional.

General Course and Stages.—Both the general and the focal symptoms of encephalic tumor vary considerably according to the stage of the disease. Early recognition of the meaning of the symptoms is of the greatest importance. With regard to the course and duration, the clinical history of encephalic tumors may, as a rule, be divided into an initial or early stage, a stage of the fully developed disease, and a terminal period. Headache, vertigo, and vomiting, slight mental changes, and disturbances of vision with the formative changes of optic neuritis, are among the first recognizable symptoms, and these are present with increased severity and greater constancy in the fully developed disease. The focal symptoms then become more definite and pronounced. Paresis deepens and passes into paralysis; spasms and contractures develop; and hemianesthesia and special disorders of hearing, taste, smell, and temperature show themselves according to the position and extension of the growth. The neuroretinitis may now be of the most phenomenal character, the papillæ being enormously swollen. In the terminal period the condition of the patient is often most distressing. The symptoms indicating destruction of the brain tissue have increased until the patient is often not only helpless and bedridden but may also be deaf and blind. Evacuations are involuntary, and loss of mental power may have advanced to almost complete dementia. Headache may persist, although in some instances, and especially in syphilitic cases, it abates or disappears with the onset of the paralysis or after the paralysis has reached a certain intensity,—probably because the intracranial tension is relieved with the breaking down of the brain tissue around the growth. As already stated, when considering brain pressure, one cause of headache is stretching of the dura, and this tension is relieved when the tissue surrounding the tumor softens. The patient may suffer less from head pains also because of his changed mental condition. In the majority of cases the general precede the focal symptoms, but occasionally this order is reversed, and some motor, visual, or auditory or other focal manifestations may appear first. Occasionally brain tumors progress with great rapidity, more frequently with fits or spurts of rapid progress. Either spontaneously or under the influence of rest, care, and good hygiene, or under the use of such remedies as the iodides, bromides, and ergot, remissions may take place in the severe symptoms of intracranial tumor. Under active treatment, especially in syphilitic cases, the symptoms may subside, and a cure, or at least an approximate cure, may be achieved; or, again, in exceptional cases the tumor may be successfully removed by surgical operation. Rare cases occur in which after attaining a certain intensity the symptoms subside and the patient continues in a quiescent and comparatively comfortable state for years.

Termination and Mode of Death.—Brain tumor cases terminate

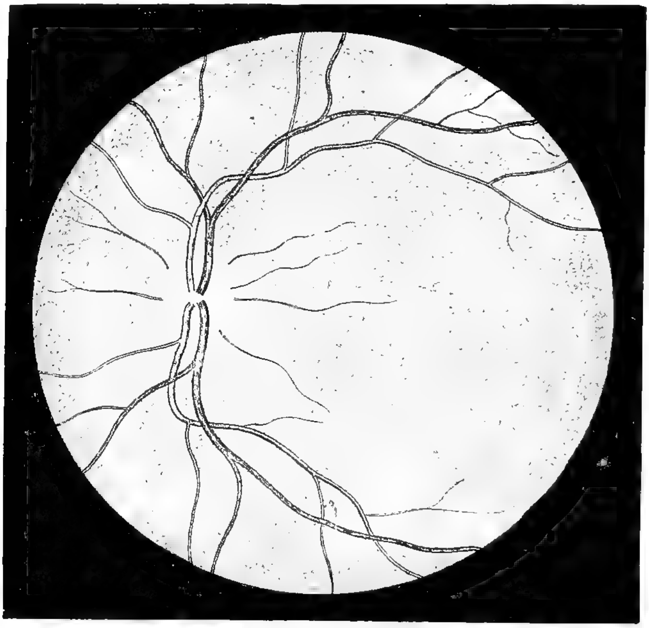
by apoplexies, by intercurrent diseases, by general exhaustion, by the effects of brain pressure exerted on the oblongata, and occasionally in other ways, as from septicemia developing from an abscess which arises adjacent to the tumor. In the majority of cases death comes on slowly at the end of a protracted terminal stage, but in a considerable percentage of cases—usually when the disease is well advanced—the patients die suddenly. Hale White mentions fourteen cases of sudden death out of a total of thirty-one observed. My experience does not confirm this high percentage, but in a number of cases I have observed either the sudden or the very rapid oncoming of death. White mentions one case of a woman who while peeling potatoes, and another of a man who when lifted to be given a drink, suddenly fell back dead. These sudden deaths may be due to abrupt changes in the intracranial pressure and local increase of this pressure in vital regions, like the floor of the ventricle, the changes probably being brought about by the sudden shifting of the head and body. The pulse has been observed to continue beating for at least half an hour after respirations have stopped.

General Diagnosis.—The diagnosis of the existence of an intracranial tumor is, in the first place, to be made by a close consideration of the general symptoms, but it will at once be recognized that some or all of these symptoms may also be present in other affections, organic and functional. Among such organic diseases are encephalopathies due to toxic agents; chronic endarteritis with nephritis; meningitis, acute or chronic; some forms of insanity, encephalic abscess or hemorrhage, and brain syphilis which has not assumed the form of a neoplasm. Among functional affections are headache and other symptom complexes due to eyestrain, grave hysteria, and migraine. The diagnosis of brain tumor from other organic affections of the central nervous system is in each case usually to be made by a close study of a few points. The focal diagnosis has been sufficiently presented under the functions and lesions of different portions of the brain, and under focal symptoms in the present section.

Toxemias. In cases of lead encephalopathy the presence of a blue line on the gums and of lead in the urine, and the occupation and previous history of the patient, will be of most service in clearing up the diagnosis. Profound malaria may in rare instances cause severe headache, general convulsions, hemianopsia, and papillitis, but in such cases most of the general and focal symptoms of brain tumor are absent, and the history of the case will help to a decision. The psychical and general symptoms which result from lithemia or gout are occasionally mistaken for those of brain tumor. Atrophy of the optic nerves in chronic alcoholism, nicotinism, or saturnism, particularly when the patients have such accompanying symptoms as chronic headache, vomiting, and vertigo, may also wrongly lead to the diagnosis of brain tumor.

Endarteritis with Nephritis. In several instances I have known the mistake to be made of confounding chronic endarteritis associated with nephritis with tumor of the brain. In one of these cases the propriety of operation at the site of an old scar was discussed. The patient died at the close of a series of uremic convulsions, and the autopsy showed the case to be one of Bright's disease with cardiac degeneration and widespread endarteritis. The correct diagnosis was finally made in this case by an examination of the urine, by a study of the accessible vessels, and by the discovery of a typical retinitis albuminurica which at first had been regarded as ordinary optic neuritis. Headache, vomiting, vertigo, and mental changes are not infrequently present in these nephritic cases, and the convulsions occasionally assume the Jacksonian type. Headache, as a rule, is not so severe. Youth would be in favor of tumor. In Fig. 297 are shown the ordinary appearances of the fundus in albuminuric retinitis, which can be compared with those in the illustrations Figs. 294 and 295. De Schweinitz calls attention to the fact that brain tumor (usually cerebellar) may cause choked disk with a star-shaped figure of whitish color in the macular region, exactly simulating the appearances usually considered characteristic of a type of albuminuric retinitis, he having seen this in a number of cases in the Philadelphia Hospital and at the Infirmary for Nervous Diseases.

FIG. 297.



Fundus oculi in a case of albuminuric retinitis. (Bramwell.)

Meningitis. Almost any of the forms of meningitis already considered may at times need to be differentiated from tumor of the brain, or, as so many neoplasms grow from the membranes and are accompanied by some adjacent meningitis, the fact that both diseases are present in the same case may require to be determined. Usually the diagnosis is between some variety of leptomeningitis and tumor (see page 293). Subacute or chronic meningitis needs the attention of the diagnostician oftener than the acute form of inflammation. A careful study of causation, although at times misleading, may be of service. Meningitis is a result of tuberculosis, syphilis, traumatism, etc.; but tumors likewise with less frequency are traceable to the same causes. On the whole, the more focal character of the symptoms and the early presence and severity of the papillitis are among the most distinctive characteristics of tumor. In syphilitic and tuberculous subjects the diagnosis is sometimes practically impossible. In children the distinction most frequently needs to be made between tumor and tuberculous meningitis, and in adults between tumor and syphilitic meningitis. The facts that gliomata are of comparatively frequent occurrence in children, and that headache is absent in some cases of glioma, must be borne in mind. In the following table are given a few of the most useful points of distinction between encephalic tumor and tubercular meningitis:

Encephalic Tumor.	Tubercular Meningitis.
Previous history of syphilis, injury, or other cause than tuberculosis.	History of syphilis, injury, or other cause than tuberculosis absent.
Hereditary history usually wanting.	Hereditary history of tuberculosis common.
Onset generally slower, but exceptions to this rule.	Onset usually somewhat rapid, with irregularities.
Papillitis comes on earlier and is usually of higher grade.	Papillitis of later development and lower grade, or may be absent.
Definite and regular focal symptoms more common.	Focal symptoms, such as palsies, and disorders of sensation and of the special senses, irregular in type and development.
Headache usually of higher grade and more localized.	Headache more diffused and sometimes of duller character.
Local head temperature sometimes significant.	Local head temperature of little or no value.
Course more regular, and duration usually of several months or years.	Course irregular, and duration usually limited to days or weeks.
Febrile phenomena paroxysmal or wanting.	Febrile symptoms usually persistent, but irregular.

While the points given in this table will answer for many cases, for others they may fail or even be misleading. Headache, for example, may be far more severe in meningitis than in tumor, and may even be absent in the latter in rare cases, as already stated. Tumors of

the brain occasionally are rapidly fatal, and the course of a meningitis even of the tubercular variety may be protracted. Tumor and meningitis are not infrequently associated in the same case, as when a solitary tubercle or tubercular tumor is present in one part of the brain while tubercular meningitis attacks the membranes of an adjacent or a distant region. Much stress is usually laid upon the predominance of basal symptoms in meningitis, but both tubercular and syphilitic meningitis of the convexity are not uncommon, and, as already shown, the base of the brain is one of the frequent seats of tumor. Tubercular or syphilitic meningitis of the convexity may give psychical disturbances, palsies, local spasms, general convulsions, sensory disturbances, and peculiar disorders of the special senses. Tubercular meningitis of the base can be readily distinguished from cases of tumor by the fact that one cranial nerve after the other is likely to become involved in the diffusing inflammatory process.

Chronic Hydrocephalus. Hydrocephalus, with or without enlargement of the head, is a frequent accompaniment of brain tumors, and especially of cerebellar, quadrigeminal, pontile, and other growths, which may by pressure or adjacent inflammation lead to obstruction of the foramens or channels of communication between the different ventricles. It may therefore become necessary to distinguish between these secondary forms and primary hydrocephalus. In chronic hydrocephalus not associated with tumor the case usually advances more slowly, and it often dates from birth or is even prenatal in origin. Although convulsions are often present, symptoms of focal irritation are not so common as in cases of tumor. Diplegias of the spastic and ataxic forms are often present in hydrocephalus, but strictly localized or unilateral spastic and paralytic phenomena are less common. Optic neuritis and atrophy may be present in hydrocephalus, but are absent oftener than in tumor, and symptoms of meningeal irritation such as headache, vertigo, and vomiting are not nearly so frequent as in ordinary hydrocephalus. While the diagnosis between the two affections can often be made, in some cases it is exceedingly difficult, as when a cerebellar or a pontile glioma is accompanied by hydrocephalus.

Abscess. The diagnosis of tumor from abscess of the brain is important, and may be difficult. It will be more fully considered after the symptomatology of the latter disease has been discussed. A few of the points to be remembered are the frequent association of abscess with aural disease, the tendency which abscess has to remain for a considerable time latent, and the more uniform and steady progress of tumor.

Apoplexy and the Results of Apoplexy. In rare instances it is necessary to distinguish between a recent apoplexy and a brain tumor. Intercurrent hemorrhage may occur during the progress of different

forms of neoplasms, and in a number of instances such a hemorrhage has first called attention to the existence of a glioma. Cases are observed which during life present clinical features of apoplexy, but in which the autopsy shows no trace of hemorrhage, recent or remote. Kuttner reports two such cases from the practice of Ewald. In one the tumor was a large cystic glioma of the occipital lobe; in the other it was a secondary carcinoma of the left temporal lobe, the latter growth being surrounded by an extensive area of softening. In ten of eleven cases recorded by Ladame, softening had occurred around the tumor. Many similar cases have been observed by the writer and others. Acute softening may therefore be regarded as the explanation of most cases of this kind. The points of distinction in the chronic stages of those cases of cerebral hemorrhage, embolism, or thrombosis which leave conditions of paralysis and other symptoms simulating the permanent condition caused by tumors have been given in full by Lloyd and the writer. Hemorrhage has usually a precedent history of diseased kidneys, hypertrophied heart, or atheromatous bloodvessels, and occurs generally in advanced life; in embolism the previous record is of rheumatism and valvular disease of the heart, and the attack occurs at any period of life, early or late. In brain tumor the history is usually one of traumatism, of constitutional infection, or of a special predisposing diathesis; and falls upon the head are common antecedents. A history of syphilis, tuberculosis, scrofula, or cancer is present. Tumor, like embolism and unlike hemorrhage, may occur at any time of life. The severe and often agonizing headache which precedes the paralytic and other phenomena of tumor is more distinctive of the latter than of the former. Papillitis is much more likely to occur in tumor than in the other affections.

Chronic Degenerative Diseases. Occasionally a brain tumor is at first supposed to be present in cases which prove to be locomotor ataxia, disseminated sclerosis, nodular or tuberous sclerosis, syringomyelia, or some other form of degenerative disease of the encephalospinal axis. These affections are diagnosticated by a careful consideration of the history and symptoms in each case. It may occasionally be necessary in the early stages of these diseases to postpone positive diagnosis. Among the most difficult cases are those in which diffuse sclerosis of the cerebellum has occurred; but such cases are rare. The ocular, sensory, ataxic, paretic, tendon, and muscle phenomena must all be carefully studied and properly referred.

Insanity. The diagnosis of brain tumor from some of the forms of insanity, such as paretic dementia, mania, and melancholia, may now and then present difficulties. The history of the onset and progress of the case, and the development of the more or less typical psychical and mental symptoms of paretic dementia, will be most

helpful in differentiating this affection. Pierce Clark has reported two cases of tumors of the brain—one of multiple gummata and the other of multiple sarcomata—in which the diagnosis of dementia paralytica had been made, and the writer and his colleagues of the Neurological Staff of the Philadelphia Hospital have in a number of instances been confronted by cases presenting the same problem of diagnosis. The subject will receive fuller consideration when general paralysis of the insane and so-called syphilitic pseudoparesis are discussed. Usually delusions of grandeur are absent in these cases, but what adds to the difficulty is the fact that in some cases of true paralytic dementia the patient is depressed, or does not present mental exaltation. In a few cases of brain tumor the patients have outbursts of acute mania or frenzy, but in true mania the general and focal symptoms of tumor are nearly always absent, although, according to Lautenbach, optic neuritis occurs in a high percentage of cases. The delusions of melancholics may lead them to believe almost anything with reference to themselves: they insist at times that they have tumors or abscesses in their brains. No difficulty need arise in uncomplicated cases.

Headache. It is not improbable that the headache of brain tumor and that of at least some cases of migraine have the same intracranial mechanism. Both may be caused by distention and irritation of the dura. In most cases, however, the diagnosis of these affections is not difficult. In migraine, symptoms absolutely distinctive of tumor, such as neuroretinitis and such focal phenomena as palsy or spasm, are not present. It must be remembered that in migraine hemianopsia, photopsia, fortification lines, and aural, gustatory, olfactory, and even aphasic disturbances may be temporarily present. Migraine is usually hereditary, and its victims have longer or shorter intervals of entire freedom from symptoms. The most difficulty will be presented when a tumor in the brain develops in a sufferer from migraine, a possibility which should be always borne in mind.

Eyestrain. In the anemic, or in those in whom from any cause the general health is impaired, eyestrain may give rise not only to headache more or less severe, but also to conditions of the optic disk and retina which are much like optic neuritis of organic origin. Even genuine and serious optic neuritis, with consequent atrophy and blindness, is said by high authorities to be due in rare instances to eyestrain; but my own experience and that of my ophthalmological friends would lead me to doubt the correctness of this opinion. It is more likely that eyestrain in these cases is associated with some local intracranial mischief or constitutional disease. The diagnosis of eyestrain from brain tumor is made by a study of the history of the case, by exclusion, and by a careful consideration of the ophthalmoscopic appearances.

Hysteria. Hysteria is frequently present in organic nervous diseases, and especially in those which, like brain tumor, cause great suffering. A decision in these cases is largely a question of keen perception and close analysis of symptoms. Double optic neuritis is of great value in deciding in favor of tumor, but occasionally conditions of the fundus are present in hysteria which are very similar to those of a low grade neuritis. In one of my own cases of grave hysteria an ophthalmologist's report upon the eyes led to an erroneous diagnosis. The fact that in grave hysteria such symptoms as monoplegia, hemianesthesia, amblyopia, and even hemianopsia and severe hystero-epileptic convulsions may be present adds to the difficulties of diagnosis.

Pathological Diagnosis.—Although the diagnosis of the pathological nature of a neoplasm in the brain is often difficult, it may sometimes be made with reasonable certainty. When considering the pathological varieties of tumor, most of the facts necessary to be borne in mind in making this differentiation were given. The determination of the nature of such growths has become much more important since surgical procedures have been adopted for the relief of brain tumors. A history of syphilis will of course be in favor of gummata; but it must be remembered on the one hand that such history cannot always be obtained, and on the other that tubercular, sarcomatous, and other neoplasms are occasionally found in syphilitic subjects. Evidences of carcinoma, sarcoma, or tuberculosis in other parts of the body than the brain may point to the correct pathological diagnosis. Tumors in young children are most frequently tubercular, although gliomata are not uncommon. Syphilitic growths are, of course, common in adults, and carcinomata beyond middle life. The effects of mercury and of the iodides may indicate that a growth is syphilitic, and yet favorable results are sometimes obtained from the use of these remedies in other cerebral tumors. If the situation of the growth can be determined, its pathological nature may be suggested. Tuberculous and gliomatous tumors are common in the cerebellum and the pons. Tumors of the surface are most frequently gummata, sarcomata, or tubercular deposits; while the centrum ovale and the callosum are usually attacked by gliomata or sarcomata. At the base a tumor may be of almost any variety.

Prognosis.—The prognosis is unfavorable. Recoveries even in syphilitic cases are rare. Bramwell states that he has come across the remains of only one old cured tumor in at least a thousand autopsies, excluding from this enumeration old syphilitic lesions. Even in syphilitic cases the growths are, as a rule, only partially removed, the connective tissue elements remaining. Tubercular and other growths sometimes become encapsulated and cease to grow or to do much harm. In rare instances intracranial tumors may be

entirely removed by operation, but, with the exception of fibromata, old inert gummata, and probably some cases of isolated tubercular growths, the neoplasms are likely to reappear. Uncertainty as to the size of a growth will often throw doubt upon the prognosis when operation is in question.

Medical Treatment.—*Remedies to relieve Particular Symptoms.* The agonizing headache of tumor of the brain sometimes can be relieved only by large doses of analgesics and sedatives. The suffering may be so great that if unrelieved the patient may have a maniacal attack or be driven to suicide. Opium and its salts are needed, the most valuable preparations being the salts of morphine and of codeine. Codeine sulphate or phosphate can be given, beginning with an average dose, say one grain, and rapidly increasing it if the exigencies of the case demand. Morphine sulphate or muriate may also be used in full doses. Hypodermatic injection is the most prompt and efficient method, but, when once resorted to, usually has to be continued. Next to morphine or codeine, phenacetin is the most efficient remedy: in some instances it is even preferable to those drugs. It can be given in doses ranging from five to fifteen grains: the effect upon the patient must be studied when its administration is begun. Large doses will generally be tolerated, but occasionally the depressing effects of the drug are markedly shown. Caffeine or strychnine may be used with it to counteract these effects. Acetanilid and antipyrin may be used instead of phenacetin. The former can be given in doses of from five to ten grains, and antipyrin in doses of from ten to twenty grains. Cannabis indica can be administered, and is most efficient when combined with the bromides and chloral, neither of which will serve to relieve the pain when given alone, but they are valuable adjuvants to the true analgesics. Either very hot or very cold applications to the head, and especially the former, may prove soothing. For the insomnia the pain-relieving remedies are of course demanded, but with them may be given special hypnotics, such as chloralamid and trional. Chloralamid in doses of about thirty grains has proved particularly useful in some of my cases. The papillitis or optic neuritis present is benefited most by treatment directed to the absorption of the tumor and the relief of intracranial tension. Horsley has shown that a remarkable improvement in the optic neuritis is sometimes produced by merely opening the skull, the swelling of the disk greatly subsiding. The eyes may need to be protected from bright glares, or even from the ordinary light; although it is remarkable that some patients suffering from double optic neuritis of high grade are unaffected by light. For the vertigo, nausea, and vomiting, when clearly due to the intracranial irritation caused by the tumor, the measures employed for the relief of headache are in the main useful. Large doses of the bromides either with or without chloral are most service-

able for the relief of spasms, local or general. Neither elevation nor depression of temperature of moderate degree calls for special treatment, but when great elevation is present, phenacetin, antifebrin, or antipyrin may prove useful in reducing it, while at the same time the pain in the head and the general irritability of the patient are relieved. Sponging with tepid water will occasionally be found comforting to the patient. Leeches to the temples or to the mastoid, wet or dry cups, counterirritants to the back of the neck, and derivatives to the limbs may do something towards mitigating the intense suffering. In rare cases, especially when sthenic and congestive symptoms are present, venesection should be employed. Cystitis, paralysis of the bowels, bedsores, and other complications must receive careful attention.

Remedies to promote Absorption and relieve Inflammation. When a gumma is suspected, mercury and iodide of potassium or of sodium should be promptly used, after the methods described under general therapeutics and under intracranial syphilis. In order of preference are mercurial inunction, with or without iodides, frequently repeated doses of calomel or of biniodide of mercury, and the iodide either of potassium or of sodium alone. Just how long to continue such treatment before resorting to operation or giving up the case as hopeless is often difficult to decide. Most patients should be given an active medicinal treatment for at least two or three months. Syphilitic growths are not the only forms of neoplasm which respond favorably to the iodide treatment. Sarcomata, gliomata, and fibromata have been known to make considerable improvement under their use. No cures, however, have been accomplished in these cases, and even in syphilitic cases only an approximate cure may be obtained. The most recent products of the infection are removed and adjacent meningitis is relieved, but older portions of the neoplasm which have become inert remain. Even when much relief and apparent cure have been obtained by the use of specific remedies, these should be resorted to again and again at intervals of two or three months for a period of at least two years.

Surgical Treatment.—A small percentage of cases of intracranial tumor can be successfully removed by trephining and ablation or enucleation. Fibromata and osteofibromata are among the most favorable cases for operation, but these include only a small percentage of the entire number. Isolated and encapsulated sarcomata are also comparatively favorable cases for operation ; and gummata which have resisted specific treatment, and tubercular growths which are apparently isolated and unassociated with tuberculosis in other organs, may both call for operation. Before operating, the exact position of the growth, that it is in an accessible area, and that it is probably solitary, should be decided. Infiltrating tumors and those secondary to disease elsewhere do not call for operation

unless it is simply to relieve the sufferings of the patient. Operation to relieve intracranial pressure and thereby mitigate extreme suffering is justifiable if its object is explained to the patient and a false hope of cure is not held out. While surgical interference should not be hastily resorted to, on the other hand it is often too long postponed. Occasionally the question of operating a second time on a case of brain tumor may be presented, either because the tumor has not been properly located by the first operation or because it has been only partially removed and the trouble recurs again at the primary site of the lesion. Such an operation is sometimes justifiable to afford temporary relief, or because of additional light as to focal diagnosis. When the situation of small cysts can be fixed, they may be opened and their cicatrization favored. Large porencephalic cysts cannot respond favorably to operation, as reaccumulation of the fluid takes place.

ENCEPHALITIS.

Encephalitis or cerebritis is an inflammation of the substance of the brain. In times not very remote, encephalitis and softening of the brain were regarded as practically synonymous, the profession in those days being largely influenced by the teachings of Durand-Fardel and his disciples. Acute inflammation of the brain was then regarded as the chief or only cause of acute softening, and chronic inflammation of chronic softening; but after embolism and thrombosis were understood, these views of the relationship of encephalitis and softening were seen to need revision. Hughlings Jackson was one of the first to show clearly that brain softening is usually a local process due to occlusion of vessels,—a necrosis rather than a result of inflammation. Similar views are now claiming more attention in connection with disease of the spinal cord. Much that has been classed as myelitis is necrotic softening, and it is quite probable that nerve trunks are at times the seat of noninflammatory necrotic processes. Nonsuppurative encephalitis has a real existence, and may be of several varieties. It is also true that it may give rise to processes of disintegration and softening on the one hand, or of hardening and atrophy on the other. In these days of frequent surgical procedure for intracranial affections, a knowledge of encephalitis may occasionally prevent serious mistakes.

Varieties.—Encephalitis, like inflammation of other organs, may be either parenchymatous or interstitial, but in most cases both parenchyma and connective tissue are consecutively or conjointly implicated. It may be either *acute* or *chronic*, the acute variety with our present knowledge being of the most clinical importance. It may also be *primary* or *secondary* and *suppurative* or *nonsuppurative*. The secondary forms are those due to metastasis, or those associated with or consecutive to other lesions. Tumors of the brain, hemor-

rhagic apoplexies, injuries of the skull or brain, and even emboli or infarcts, may have surrounding zones of secondary meningitis and encephalitis or meningoencephalitis. Our concern will first be with the acute and chronic forms of primary encephalitis. Acute primary encephalitis may be either *focal*, *diffuse*, or *disseminated;* chronic primary encephalitis is usually diffuse, but some forms of it are localized to lobes or lobules. Acute focal encephalitis has been described as of different forms according to its location—in the cortex, subcortex, pons, or oblongata. *Cortical polioencephalitis* was suggested by Strümpell in 1884. While acute primary encephalitis may be limited to the gray matter of the brain, and thus be entitled to the designation polioencephalitis, a number of cases have been reported which show that this disease may attack either limited or comparatively large areas and may in its immediate effects involve both gray and white matter and both parenchymatous and interstitial tissues. It is best, therefore, to discuss simply primary acute focal encephalitis, leaving the question of its polioencephalitic character in some cases unsettled. Cases of focal encephalitis, usually hemorrhagic, and similar in their pathological findings, have been observed in all portions of the brain. The terms which have been introduced in describing some of the best known, or at least the most frequently discussed, of these forms are certainly calculated to mislead, a remark which applies not only to Strümpell's polioencephalitis, but also to the *polioencephalitis superior* and the *polioencephalitis inferior* of Wernicke. Polioencephalitis superior is described by Wernicke as an affection of abrupt origin which attacks respectively the nuclei and adjacent fibres of the aqueduct and preoblongata,—structures chiefly related to the ocular nerves and muscles. Some form of ophthalmoplegia is the predominating clinical feature. In polioencephalitis inferior the nuclei and other structures of the postoblongata are attacked, giving labioglossolaryngeal paralysis with or without respiratory and cardiac involvement. These affections will need to be considered again under diseases of the pons, oblongata, and cranial nerves. The names used above to designate them have become so engrafted into the literature of the subject that it is better to make use of them in discussing encephalic inflammation, at least as synonyms. The terms "superior" and "inferior" are, however, misleading, as they might just as well be applied to cortical and subcortical focal encephalitis, or to cerebral and subcerebral varieties of the disease. A general encephalitis is clinically unknown, for the simple reason that death must ensue before the inflammation involves the entire brain; but forms of *diffuse* inflammation, both acute and chronic, are well observed clinical and pathological types. A lobe or almost an entire hemisphere may be involved in the inflammatory process, which is usually subcortical, but may be conjointly cortical and subcortical. A diffuse *periencephalitis* or inflammation

limited to the surface of the brain may be almost general, and the soft membranes of the brain are often implicated in such inflammation, as would be expected from the intimate connection between the arachnopia and the brain. This associated inflammation of membranes and brain surface constitutes a *meningoencephalitis.* In paralytic dementia or general paralysis of the insane, periencephalitis or meningoencephalitis is present with other lesions. *Disseminated encephalitis* has been described, and may be of a widely distributed miliary form, or may occur in patches of varying size. Etiological varieties of encephalitis, such as the luetic, alcoholic, saturnine, puerperal, and postfebrile, are sometimes recognized, but these need here only this general reference.

ACUTE FOCAL ENCEPHALITIS.

As stated in the last paragraph, Strümpell at first contended for the existence of a form of localized cortical polioencephalitis, analogous in causation and nature to poliomyelitis and the bulbar types of polioencephalitis of Wernicke. Much opposition has been shown to this suggestion, and some writers have made sweeping assertions denying even the possibility of its occurrence. I can see no reason why an inflammatory process due to infection or other cause might not attack the gray matter of the cerebrum if it attacks that of the bulb or the spinal cord; but the weight of recent observations is in favor of the view that the inflammatory lesions, as a rule, are not strictly confined to the gray matter, although they may be so limited in rare instances. In support of Strümpell's original view, Moebius has given the history of two children in one family who were stricken down with fever, one of whom developed a typical poliomyelitic paralysis of the upper extremity, and the other a spastic hemiplegia. Strümpell has recorded cases of apoplexy in adults simulating closely in their clinical history attacks of embolism, but revealing on autopsy hemorrhagic encephalitis of both gray and white matter. Other observers have reported similar cases, some of them having been observed during the recent epidemics of influenza. Schmidt has recorded one such case in which the autopsy revealed in the head of the left caudatum and lenticula an isolated mass of bloody detritus, and in other localities, on both sides of the brain, hemorrhagic fissures and punctiform hemorrhages. Fürbringer and Koenigsdorf have corroborated the existence of this affection. The term acute focal encephalitis is to be preferred to cortical polioencephalitis, as the lesions have been found to affect both gray and white matter. It is a disease both of adults and of children, and it is not improbable, as has been held, that it is the cause of some of the cases of infantile cerebral paralysis, both hemiplegic and diplegic. At the time of the acute symptoms the patient may be supposed to be suffering from typhoid fever, malarial fever, influenza, or some other

form of infectious disease; or the encephalitis may originate in the course of such febrile affections.

Clinical History.—The disease as observed in young children usually comes on acutely, vomiting, convulsions, and fever being prominent symptoms. Headache, irritability, vertigo, and drowsiness may be present for a few days. The child then passes into an apathetic or comatose state, which may alternate with periods of restlessness or even of delirium. Fever of varying grade is present; chills may or may not be among the manifestations; tetanoid symptoms and opisthotonos may be present early, or may be entirely absent. If cranial nerve symptoms are present, the probabilities are that the encephalitis has become diffuse, or that basal structures are coincidently involved with the cortex. In most severe cases the mental and physical conditions become aggravated, respiration is interfered with, Cheyne-Stokes breathing may be present, and the patient dies in a week or two; but the acute symptoms not infrequently pass away, leaving residual affections of more or less severe type. Paralyses, contractures, atrophies, monochoreas or hemichoreas, hemiathetosis, chronic convulsions, often of the unilateral type, and a greater or less grade of imbecility, are the most persistent of these residual affections. The duration of the disease as an acute affection may vary from a few days to several weeks. Remissions and exacerbations in the acute symptoms are observed in some cases. Few children who are attacked with the disease pass scathless through it; something in the form of paralysis, with or without spastic phenomena, is generally left behind. When acute focal encephalitis attacks the adult brain, the symptoms do not differ markedly from those observed in the young. Irritative phenomena, as headache, fever, and convulsions, are on the whole less severe, and the residual paralyses, contractures, and other conditions may be less extensive. When the inflammation attacks parts chiefly below the cortex, the focal symptoms will of course differ with the location of the lesion. More or less localized forms have been recorded in the centrum ovale, basal ganglia, and capsules. A sharp distinction cannot always be made between focal and diffuse encephalitis. Probably at first the inflammation is more or less diffuse in most cases, but its destructive effects become concentrated in small areas, the disease in this way having striking analogies to the so-called poliomyelitis, which at first is often a diffuse myelitis. The symptomatology may therefore at first be that of a diffuse inflammation, the focal manifestations becoming evident as the acute stage subsides.

Etiology.—Acute focal encephalitis usually arises after or during the course of some acute infectious disease, as influenza, typhoid fever, diphtheria, or the eruptive fevers. Like anterior poliomyelitis, it would appear at times to have an infection peculiarly its own; that is, none of the known infectious diseases are recognized

as present. Syphilis, alcoholism, and traumatism are occasional causes of both cortical and subcortical focal inflammations, but more frequently they give rise to diffuse encephalitis.

Pathological Anatomy.—In a few recorded cases with autopsies, circumscribed and usually small areas of softened tissue, which have a red appearance due to punctiform or capillary hemorrhages, have been found. The microscope shows distended and ruptured vessels, leucocytes, and granular cells. Neuroglia cells are proliferated, and the destruction of true nerve elements is greater or less according to the extent and intensity of the pathological process. Whether the inflammation in infectious cases is the direct or indirect result of the infecting microbes has not yet been determined. In some instances comparatively large hemorrhagic and broken down areas are present.

Diagnosis.—As in poliomyelitis, the patient in the acute stage of this disease is often supposed to be suffering simply from an ephemeral, intermittent, remittent, typhoid, or other fever, the presence of the encephalitis not being recognized. Attention to the cerebral symptoms, and to the progress of the disease and its effects, is necessary to make the diagnosis clear. Often the true nature of the affection is not recognized until after several weeks, when a palsy of the limbs or of the face is discovered. The patient is often supposed to be suffering from one of the forms of meningitis, and it is difficult to separate the two affections. The presence of rheumatism and endocarditis, and the abrupt origin of the paralysis, will assist in making the diagnosis from embolism. When the cerebral and spinal palsies of children are considered, the methods of differentiating the residual affections of focal encephalitis will claim attention.

Prognosis.—The prognosis as to the acute attack varies, and is dependent largely on the virulence of the infection which leads to the encephalitis. If the issue is fatal, death may be due not directly to the encephalitis, but to the general toxemia of which the encephalitis is one of the serious results. The prognosis as to residual affections also varies. Occasionally patients recover completely from a paralysis. In a larger number of cases partial recovery takes place, the patient being left more or less monoplegic, hemiplegic, or diplegic. Some improvement may continue for several months after the acute attack.

Treatment.—Little need be said as to treatment. Often, the focal encephalitis not being suspected, treatment is simply directed to the acute febrile disorder from which the patient is suffering or supposed to be suffering. When encephalitis is suspected, the treatment should be similar to that of acute meningitis,—absolute quiet, sedatives, analgesics, cold applications to the head, and the administration of purgatives, especially of calomel or other mercurials.

ACUTE DIFFUSE NONSUPPURATIVE ENCEPHALITIS.

Diffuse encephalitis may be *acute, subacute,* or *chronic.* A case beginning acutely may soon terminate fatally, or before death or partial recovery takes place it may be so prolonged as to become either subacute or chronic. A clear separation, therefore, cannot always be made between these forms. Probably the term subacute is most applicable in the majority of cases. Diffuse encephalitis may also be *nonsuppurative* or *suppurative;* and in this section attention will be directed to the former. As already stated, a sharp distinction cannot always be made between focal and diffuse encephalitis; but a few recorded cases clearly show that a severe inflammation sometimes involves large continuous areas of the interior as well as of the surface of the brain, leaving effects that can be referred only to extensive destruction and degeneration. Either softening or hardening of the brain substance may be the ultimate result of diffuse encephalitis, softening being more likely to follow an acute or a subacute inflammation, and hardening a chronic inflammation. In some extensive and severe cases the affected areas are reduced to a semipurulent mass. When the subject of tumors of the brain was under consideration, it was stated that, properly speaking, both gummata and tubercular tumors are acknowledged to be the result of inflammation, but for clinical reasons they were regarded as tumors, the products of the inflammation being so aggregated and so isolated as to give definite focal symptoms. A few special words may here be said about syphilitic encephalitis. The syphilitic virus acts chiefly on the bloodvessels, lymphatics, and connective tissue. Parenchymatous lesions, whether cerebral or spinal, are, as a rule, secondary. The tissues react to the irritant much as in other inflammations. The products of the syphilitic inflammation may be a true gumma or a diffuse infiltration, the latter being so distributed as to constitute a diffuse or disseminated encephalitis. Recent pathological investigations of syphilitic and tuberculous myelitis throw considerable light upon the nature of all forms of inflammation of the central nervous system. Any sufficiently virulent infection or toxic agent, or a serious traumatism, may produce inflammatory and necrotic changes similar to those which have been demonstrated as occurring in the spinal cord and brain, especially in the former, as the result of syphilis. In central syphilitic disease the process is a diffuse and irregular one, giving a certain stamp to most cases of specific origin. Whatever be the cause of the inflammation, the parenchyma, although it is rarely primarily involved, is always sooner or later implicated in the inflammation, or becomes the seat of degeneration through deprivation of nourishment. The softening of the brain in all forms of encephalitis is usually in reality necrotic, at least so far as the parenchyma is concerned, being due to obliterative inflammation of the vessels or to

their occlusion by exudates or growths. So far as the noble elements of the brain are concerned, the softening or degeneration is the result of inflammation rather than inflammation itself; but the interstitial and parenchymatous tissue becomes so blended and disintegrated in the complicated morbid process that the whole is best regarded as a diffuse inflammatory lesion. Because of its comparatively distinctive features, and for other practical reasons, syphilitic encephalitis will be treated at greater length when intracranial syphilis is separately considered. The following brief discussion of diffuse nonsuppurative encephalitis is concerned chiefly with nonsyphilitic forms of the disease.

Clinical History.—The character and succession of symptoms in a case of acute or subacute diffuse encephalitis differ according to the location and extent of the inflammation.* Certain symptoms are likely to be present in all cases, and among these are headache, commonly dull and deep seated. mental apathy or torpor. fever with delirium. and localized or diffused spasms. The mental apathy or torpor is often present even in acute cases when a large part of the brain is involved, and in almost all cases if the patient survives for a considerable time. After a severe injury, or from some unknown cause, probably infection. the patient begins to have spasms and twitchings without loss of consciousness. Paralysis with spasticity, and mental deterioration, become pronounced. Sensation may remain normal, or may be involved according to the site of the lesion. Reflexes are commonly increased on the paralyzed side. The clinical history of the acute or subacute stages of such a case is similar to that of a considerable percentage of cases of spastic infantile paralysis with local spasms or convulsions. A case is reported by Sharkey which on gross examination showed only general enlargement of the affected hemisphere with some softening of its interior, but in which the microscope revealed a severe and widely distributed encephalitis. This patient, who was thirty-nine years old, presented a train of severe symptoms. and died about four weeks after their onset. At first he complained of some twitching in his right hand when writing, and of numbness on the inner side of the right cheek. Taste on the same side was abolished. He had a series of fits which left him in a nearly unconscious condition. The right pupil was larger than the left, and neither responded to light. Sensation appeared to be absent, and paralysis was present on both sides, but was much more complete on the right than on the left. The superficial reflexes were absent, but knee jerks were normal and equal. He had a number of convulsions. and from time to time various local spastic phenomena,

* In Brain, vol. xvi., 1893, pages 213–229, Knaggs and Brown have made a valuable contribution to the subject of diffuse encephalitis, which I have freely used in the preparation of this section. The report of the case is accompanied by details of an autopsy and microscopical examination.

such as trismus, turning of the eyes, and incessant twitchings of the muscles of the face, tongue, jaw, and neck. The temperature during the acute or subacute stages varies considerably, usually at first being moderately elevated; but during the progress of the affection it is more likely to be subnormal or to range close to the normal. When one hemisphere is affected, the temperature has been found in a few cases to be subnormal on the side of the paralysis,—that is, the side opposite to the lesion. The pulse, like the temperature, is often subnormal, but varies according to the stage or intensity of the process. Involuntary evacuations, as a rule, are present. Optic neuritis is not so common as in meningitis or purulent encephalitis, and it may be unilateral.

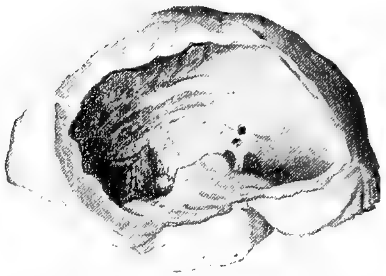
Duration and Course.—The duration is usually from twenty-four hours to four or five weeks. A few cases are rapidly fatal; the majority linger for several weeks, and some become chronic. A very few cases have been recorded in which acute or at least active symptoms persisted for several months with remissions and exacerbations. The course of the disease, even when short, is usually somewhat irregular, symptoms of irritation taking the lead at one time, and states of apathy and torpor predominating at another.

Etiology.—The disease, except when syphilitic or alcoholic, usually occurs in infancy and childhood; the cases collected by Knaggs and Brown were all under twenty years. Inherited weaknesses of various kinds act as predisposing causes, and traumatism is the most frequent exciting cause. Alcoholism, or any infectious disease or toxic agent, is a possible cause. Both diffuse and focal encephalitis have been recorded as occurring during epidemics of influenza.

Pathological Anatomy.—In the early stages of diffuse inflammation of the brain, vessels are distended and dilated, and capillary and arteriole extravasations probably take place. In cases of severe type in which autopsies have been made, after the inflammation has existed for a comparatively short period, softening of the brain substance has been the chief pathological feature. In the more chronic cases induration has been found. The changes may involve nearly an entire hemisphere or extensive areas of both hemispheres, or they may be more restricted. The right hemisphere which was the seat of widespread inflammation in the case reported by Knaggs and Brown was found at the autopsy fluctuating and tremulous to the touch. The ventricles and their horns were enormously dilated. Careful examination of the brain after hardening showed the lining membranes of the ventricles much thickened, and the right striatum and other parts of the hemicerebrum sclerosed and greatly atrophied. The left lateral ventricle and its horns were also dilated, but not nearly to the same extent as on the right. The inflammation had been limited to the right hemisphere, but dilatation of the connected ventricu-

lar cavities had resulted, probably because of closure of the iter from obliterative inflammation. In Figs. 298 and 299 are shown the appearances of the ventricles and hemispheres from sections and drawings made by Dr. Brown after the specimens had been hardened. In some cases, perhaps in the majority, evidences of more or less extensive inflammation are seen in the membranes. Sometimes, although extensive inflammation of the brain substance has been present, the appearances to the naked eye are not such as to enable a positive opinion to be given. In Sharkey's case, for example, the macro-

Fig. 298.



Longitudinal section of right hemicerebrum (after hardening), showing the appearances of the enlarged lateral ventricle and the surrounding diseased areas, in a case of subacute diffuse nonsuppurative encephalitis following traumatism. (Knaggs and Brown.)

Fig. 299.

Longitudinal section of left hemicerebrum (after hardening), showing enlarged lateral ventricle, but without surrounding inflammatory and degenerative changes; same case as Fig. 298. (Knaggs and Brown.)

scopic postmortem examination was regarded as practically negative, but the microscope showed dilated vessels, leucocytes, and other evidences of true inflammation. Microscopically all traces of nerve structure may be lost in the parts which have been the seat of the most active inflammation, and the neuroglia is condensed and infiltrated with inflammatory products. Perivascular spaces are enlarged. Descending degeneration may be found in cases which have survived the inflammation for a short time.

Diagnosis.—It is certainly difficult, with our present knowledge of the subject, to distinguish between some cases of diffuse meningitis or meningoencephalitis and diffuse subcortical encephalitis. A symptom which was present in all the cases collected by Knaggs and Brown, and which is therefore of diagnostic importance, is the state of vacuity and helplessness in which the patients lay for long periods. These writers believe that long continued torpor of mind and body is the most salient feature of chronic diffuse encephalitis; but, as this condition may not develop in acute cases of brief duration, its diagnostic value for such cases is impaired. Even in the acute cases, when the brain is very extensively involved, the torpor is present, and the symptoms are those which show involvement of large subcortical areas. The symptoms as detailed in the clinical history are

largely the same as those given under focal encephalitis, the focal cases being at first more or less diffuse, or the diffuse cases in other instances having at first been focal. In the diffuse form of inflammation, especially when it is largely subcortical, states of vacuity and torpor and symptoms which show dissociation of cerebral faculties are more prominent. In focal cases the acute stage is briefer and the residual conditions, while very definite and distinctive, are not so serious. After a focal encephalitis of limited extent, only moderate monoplegia or hemiplegia, with little or no mental deterioration, is left. When diffuse encephalitis does not result fatally, it always leaves severe and extensive residual affections.

Prognosis.—The prognosis as to complete recovery is always bad. When the diffuse inflammation attacks both hemispheres, and when it is due to a very severe traumatism or to a virulent infection, death may occur in a few days. In other cases the patient lives for several weeks or months, and then dies from exhaustion or lingers for years deteriorated in mind and a sufferer from some one of the forms of hemiplegia or diplegia. According to Oppenheim, in acute nonsuppurative encephalitis, very abrupt onset and severe symptoms, including high temperature, are signs of danger, while a slow onset, low temperature, and protracted course are of good omen. The cases referred to by Oppenheim were probably more focal than diffuse, but the remarks apply equally to both varieties.

Treatment.—During the acute stage the treatment should be that employed for other forms of acute inflammation,—mercury, iodides, bromides, ergot, local and in rare cases general bloodletting, and ice bags to the head. Absolute rest should be enforced. Special symptoms should receive attention. Convulsions, for instance, may call for the use of the bromides, antipyrin, antifebrin, sulphonal, or similar remedies.

DISSEMINATED ENCEPHALITIS.

While disseminated encephalitis of several forms has been described by different authors, no well defined clinical type of this affection is known. Occasionally disseminated encephalitis, like disseminated myelitis, is an acute affection; and such cases so far as known are due to syphilis or to some of the well known infectious diseases. Various acute diseases of infectious or contagious character, as diphtheria, scarlet fever, variola, influenza, typhoid or typhus fever, and erysipelas, may set up a disseminated encephalitis of peculiar character. Little foci of inflammation are scattered here and there throughout the brain. This form of disseminated encephalitis may also be present in septicemia. What has been termed mycosis of the brain is a form of disseminated inflammation,—an affection in which little colonies of micrococci are found scattered throughout the brain. Charcot and Gombault studied and described a form of cerebrospinal syphilitic inflammation which is chronic and results in

the formation of sclerotic patches, some of which undergo caseation. The lesions of this disease are more frequently spinal than encephalic, but they may be confined to the encephalon. Doubtless nodose periarteritis and some of the forms of chronic syphilitic meningitis are allied to and associated with this variety of disseminated encephalitis. Danillo has described a form of miliary encephalitis in newborn children due to septic causes, such as suppuration of the umbilicus. In some cases, at least, insular or disseminated sclerosis of the brain is of inflammatory origin. Some forms of focal sclerosis of the nerve centres are due to syphilis. According to Lancereaux, the syphilitic forms of sclerosis can be diagnosticated from the nonsyphilitic by the greater tendency of the neuroglia to fatty degenerations, and by the occurrence of foci of softening in their neighborhood. The symptomatology of disseminated encephalitis, acute or chronic, must of course be very irregular and variable in character, depending upon the number, size, and location of the points or patches of inflammation. No symptom picture of the acute cases can well be given. It may be as multiform and irregular as the lesions. Paresis, anesthesias, amnesias, cranial nerve disorders of slight or marked character, and visual, auditory, and other phenomena of the special senses, may be among the symptoms.

CHRONIC ENCEPHALITIS.

The forms of focal, diffuse, and disseminated encephalitis which have been described usually run an acute or a subacute course, but occasionally the inflammatory stage is so prolonged as to justify the application of the term chronic. The term chronic encephalitis is, however, more commonly applied to several other forms of inflammation, although these have not yet attained a well defined position as the result of either clinical or pathological study. According to Gowers, certain rare forms of chronic softening of the brain are probably inflammatory in nature. He also believes that a chronic focal inflammation of the brain substance is sometimes observed in patients with a gouty diathesis, the symptoms being those of focal lesions of the cortex, such as convulsions beginning locally, hemiparesis, partial aphasia, delirium, and slight pyrexia. Optic neuritis is absent, and headache is not prominent, but in other respects the symptoms are like those of brain tumor. In still other rare instances cases are observed which simulate brain tumor even more closely, such symptoms as headache, vomiting, and even well marked papillitis being present. Hughlings Jackson has reported such a case in which the severe symptoms lasted for six months, and in which the microscope showed slight diffuse inflammatory changes throughout the substance of the brain. Inflammatory hypertrophy of the brain cortex has been described with a symptomatology which includes epileptiform convulsions and some arrest of mental develop-

ment. The different forms of so-called sclerosis of the brain are regarded by some authorities as primarily varieties of interstitial inflammation, and it is probable that in some instances they are inflammatory in origin, in others simply reparative and degenerative, or that the changes are due to some irritant morbid process, usually the result of infection, the process standing in some way intermediate between true inflammation and simple degeneration. It has been shown in the section on diffuse encephalitis that one of the results of an active acute inflammation is a hardening of the brain substance throughout a lobe, or it may be throughout almost an entire hemisphere; but this is to be ranked not as a chronic encephalitis, but as the sequel of an acute or a subacute encephalitis. In that form of insanity known as paralytic dementia, a more or less diffuse inflammation of the surface of the brain is one of the most constant lesions. It constitutes a periencephalitis, but the arachnopia is also inflamed and thickened, and is usually adherent in many places to the cortex, so that the inflammation presents itself rather as a meningoencephalitis. Various grades of inflammation are also present in the dura; the vessels are seriously diseased; and nerve cells and fibres are atrophied so that convolutions and even lobules or lobes may present an irregularly shrunken appearance. According to one view, the disease is primarily an interstitial inflammation beginning in the vascular apparatus, soon involving the connective tissue, and eventually by secondary processes implicating and destroying the true nerve elements. According to another view, the disease is primarily parenchymatous and noninflammatory. Nerve cells and processes first disappear, and later the vessels are diseased, and neurogliar hyperplasia takes place. A chronic meningoencephalitis, however, is present at some stage of the disease, according to either view. The encephalitis is not strictly confined to the cortex, but may invade the brain substance to various depths. Ependymitis is often present. Paralytic dementia will be considered more fully elsewhere, attention being directed to it here simply to make more complete our survey of the entire subject of encephalitis. It is necessary to bear in mind also in this connection that a meningoencephalitis or periencephalitis may be present and not result in this disease, which is probably one which is due to a toxin produced in the system usually following syphilitic infection, a general assault being made upon the entire nervous system. The periencephalitis is simply an important part of the morbid processes which are set up in the interstitial and parenchymatous tissues of brain, cord, and peripheral nerves. Raymond considers syphilitic general paralysis to be a chronic interstitial inflammation, having its point of departure particularly in the cortical capillaries, the lesion being termed by him diffuse embryonic meningoencephalitis or diffuse vascular meningoencephalitis.

DIFFUSE SUPPURATIVE ENCEPHALITIS.

Suppurative encephalitis and abscess of the brain, although usually considered together, are not strictly synonymous, as a diffuse suppurative encephalitis may be present without the formation of an abscess in the clinical sense—a localized collection of pus with definite boundaries. On the other hand, a circumscribed accumulation of pus may be found within the brain without evidences of adjacent or remote intracranial inflammation. In like manner suppurative meningitis may be present without the formation of an abscess; but an abscess may form either outside or within the brain membranes as the result of meningitis, or of purulent disease in some remote position in the cranium or elsewhere in the body. It is therefore necessary for the purposes of diagnosis and treatment to regard abscess as a focal affection, and suppurative encephalitis as either focal or diffuse and as possibly occurring without the formation of true abscess. The symptomatology of acute suppurative encephalitis is in general terms that of the nonsuppurative forms, with the addition of symptoms due to septic infection. It is chiefly that of more or less diffuse irritation and compression. Distinctive focal symptoms are wanting. Fever of irregular type is present. The temperature is sometimes below normal, but varies according to the degree and character of the involvement of the brain, sudden oscillations being comparatively common. The variations in pulse and respiration are similar to those of temperature. Chills or chilly sensations are common. Dull headache, delirium or stupor, local or general spasms, irregular paralytic phenomena, and disturbances of speech, of sensation, or of the special senses, may be present. Optic neuritis is comparatively common. Meningitis, and particularly leptomeningitis, often accompanies suppurative encephalitis. When an abscess does not form for a long period, or when the final condition is one in which the brain substance is converted into a semipurulent mass, sometimes termed a cold abscess, a few symptoms are present over a long period, the most important of which are pain, dizziness, mental confusion, torpor, athetoid or choreic movements, convulsions and gradual loss of motor power, speech, or sensation, according to the parts invaded. It is not necessary to go into special details as to the etiology, pathology, diagnosis, and treatment of those forms of suppurative encephalitis which do not result in localized collections of pus, as these will be given under abscess. Postmortem examination shows great destruction and disintegration of the nerve elements. The parts swarm with leucocytes, the brain mass being reduced in some places to a soft pulpy state, in others being fluid and semipurulent. Various pathogenic microorganisms are present. The possibility of the occurrence of suppurative encephalitis should be borne in mind in the treatment of injuries and of serious cases of infectious disease.

ENCEPHALIC ABSCESS.

An encephalic abscess is a circumscribed collection of pus anywhere within the cranial cavity; but experience shows that it is much more frequent in certain situations, as in the temporal lobe and the cerebellum, these being the most frequent locations of brain abscess, because they are so commonly associated with disease of the ear. The following summary from a table by Allport shows the most common locations of intracranial abscesses, and their relative frequency in different sites.

Location of Ninety-Eight Cases of Intracranial Abscess.

Temporal lobe	40
Parietal lobe	7
Occipital lobe	1
Frontal lobe	2
Cerebellum	31
Pons	3
Cerebellar peduncle	1
Middle cranial fossa	1
Posterior cranial fossa	2
Superior frontal convolution	1
Apex of the petrous bone	1
Beneath the dura on the outer surface of the petrous bone	3
Diffuse subdural abscess	5

Clinical History.—*Active and Latent Periods.* The symptoms of brain abscess vary according as the affection runs a chronic or an acute course. An abscess may remain encapsulated and latent, or nearly so, for months or years. Even when abscess is suspected the symptoms are often for a long time obscure. Sooner or later, in cases of chronic abscess, active terminal symptoms show themselves, and these are usually similar to those of an acute abscess which runs a somewhat rapid course from the start. Owing to these facts, the clinical history can sometimes be divided into a latent or semilatent stage and a period of active manifestations. The entire course may be sharply acute, terminating in a few weeks; or, after an active initiatory period, the symptoms may become partially or completely latent, the disease again clearly manifesting itself by slow degrees or by abrupt transition. An injury sometimes calls the acute symptoms into activity. In thirty-six cases of traumatic abscess recorded by Beck the time between injury and operation in fourteen instances was counted by weeks, by months in sixteen, and by years in six. In the latent stage, such general symptoms as mental disturbances, headache, occasional vomiting, and vertigo, may be present for a longer or shorter time, with free intervals.

General Symptoms of the Active Period. The general symptoms of the active stage of abscess are much the same as those of brain

tumor; but they vary somewhat with the location and extent of the suppurative process; also according as it is superficial and associated with meningitis or is isolated and deep seated in the substance of the brain. The most important of these general symptoms are headache, nausea or vomiting, vertigo, hebetude, and convulsions. Optic neuritis is occasionally present, but not so often as in tumor of the brain. According to Knies, the most frequent form of papillitis is what he terms "obstructive neuritis," in which the swelling is slighter but the inflammatory phenomena are more pronounced than in typical choked disk. He considers this the most characteristic ophthalmoscopic finding in abscess of the brain, it being usually bilateral, although more marked on one side. When unilateral it is generally on the same side as the abscess, which is usually located in the frontal or the temporal lobe. Delirium may be present even in uncomplicated cases, but is more marked when there is associated meningitis. At some stages the symptoms are chiefly such as indicate a suppurative process, as fever with rigors followed by sweating. When the abscess is for a long time latent, its probable presence is sometimes indicated by such irregularly recurring septic phenomena. The temperature changes usually point to septic infection rather than to a focal encephalic lesion, although at times differences in temperature may be due, in part at least, to differences in the sites of the abscesses. The temperature, after an initial rise, usually falls and remains below normal. In cases observed by Eskridge the temperature taken in the axilla was found to be from one half to three degrees higher on the paralyzed than on the unaffected side. After evacuation of the abscess it becomes nearly equal on the two sides. The pulse is usually slow, and of fair volume unless meningitis is present, when it may be both rapid and irregular. It may fall as low as forty or fifty beats in the minute, or even less. The bowels are often obstinately constipated until just before death. Inhibited or sluggish cerebral action is often a significant feature; and rapid emaciation is a symptom of considerable value, especially when it cannot be explained by diarrhea, vomiting, and fever. The pupils show many variations, so that nothing definite can be determined from their study. Local tenderness of the scalp is sometimes present, even in temporal and cerebellar cases, but it is not a symptom upon which reliance can be placed, except when indicative of mastoid disease.

Symptoms due to the Association of Encephalic Abscess with other Intracranial Affections. When leptomeningitis is associated with encephalic abscess, the focal and other symptoms of a circumscribed collection of pus may be the first to appear, and then follow symptoms of meningeal involvement. These symptoms are such as general irritability, local or diffuse spasms, high temperature without decided remissions, and rapid pulse. On the other hand, the symptoms of an active and acute purulent meningitis may mark the first

stages, and the focal and other symptoms of abscess be added; the symptom picture then becomes very confusing. Sinus thrombosis may be associated with abscess, but from the confused and extensive array of phenomena it may sometimes be possible to pick out those of both abscess and thrombosis. The symptoms of secondary thrombosis as given on page 306 should be studied. Usually when this association occurs the patient suffers from frequent rigors, the pulse becomes weak and rapid, and the temperature high with decided remissions. Some of the local evidences of thrombosis in the posterior cervical triangle, tenderness along the jugular of the affected side, and pulmonary infarcts may be present. The cranial and intracranial manifestations due to accompanying suppurative aural disease are of course important, and include such symptoms as pain in the ear, deafness, facial paralysis, tinnitus, and local changes in and around the ear. Allport has tabulated the symptoms present in one hundred and sixty-nine cases of purulent brain deposits and accumulations following ear disease. They included ninety-eight cases of abscess proper, the others being mostly cases of purulent thrombosis and meningitis. The table given below is modified from his tabular statement.

Most Important Symptoms in One Hundred and Sixty-Nine Cases of Intracranial Purulent Deposits or Accumulations.

Head pain	78	Exophthalmus	4
Nausea and vomiting	41	Diplopia	2
Vertigo	22	Strabismus	10
Optic neuritis	11	Aphasia	8
Delirium	36	Facial paresis or paralysis	28
Convulsions	22	Paresis or paralysis of limbs	19
Stupor	18	Spasms of limbs	7
Unconsciousness	17	Spasms of facial muscles	4
Coma	39	Paralysis of auditory nerve	2
Insomnia	2	Neuralgia of trigeminus	2
Somnolence	11	Opisthotonos	3
Chills	38	Ear pain	35
Temperature high	8	Deafness	11
Temperature medium	42	Tinnitus aurium	3
Temperature subnormal	2	Tympanic necrosis	6
Sudden rise and fall of temperature	2	Tympanic granulations	6
Pulse high	6	Tympanic polypus	7
Pulse medium	33	Swelling in front of ear	3
Pulse subnormal	2	Swelling over ear	4
Diarrhea	3	Swelling under ear	4
Constipation	11	Mastoid swollen and tender	35
Incontinence of urine	3	Pus in mastoid opening	16
Amblyopia or amaurosis	6	Pus not in mastoid opening	7
Pupils dilated	8	Spontaneous mastoid opening	5
Pupils contracted	8	Edema of eyelids	1
Pupils sluggish	2	Facial veins enlarged	2
Nystagmus	1	Facial edema	2
Ptosis	6	Edema of the neck	3

Symptoms due to the Association of Abscess with Disease of the Lungs or other Organs. The symptomatology of abscess of the brain may be confused not only by its frequent association with leptomeningitis, sinus thrombosis, and local affections of the ear and cranial nerves, but also because it may be complicated with symptoms due to the infectious morbid processes, and diseases which are present in other organs and some of which may be the septic sources of the brain affection. The febrile phenomena, the emaciation, and the general prostration or even typhoid state sometimes present may be dependent as much upon serious inflammatory and suppurative disease of the lungs as upon the purulent accumulation within the cranium. In a case reported by Eskridge and Parkhill, the two abscesses which were found in the brain were caused by septic emboli from an old gunshot wound in the right lung. This lung in its lower third was a solid mass of fibrous tissue, and the bronchi here and in the middle lobe presented cavities filled with mucus and pus. The lungs or other organs may be the seat of multiple abscesses. In such cases the symptoms strictly referable to the intracranial lesions must be carefully separated from those due to general septicemia or to local disease in organs other than the brain. Less reliance in these complicated cases can be placed upon peculiarities of temperature and pulse, upon rigors followed by sweats, and upon emaciation and exhaustion. Serious disease of the lungs may almost mask the brain disease.

Focal Symptoms. The focal symptoms of abscess, like those of hemorrhage, softening, and tumor, vary, of course, with the location of the abscess and with its extension and radiation from a given location. These symptoms will be readily understood by a consideration of what has already been taught under encephalic localization and other focal diseases. Abscesses of the motor cortex or subcortex are not common, but when present give varying grades of paresis or paralysis and spasms of a more or less limited character, but these are less frequent than in brain tumor, and when they are present are not likely to have such definite initial or signal symptoms. When, as occurs in rare instances, an abscess involves the internal capsule, hemiplegic and even hemianesthetic symptoms may be present, and they will vary according to the extent of the lesion. Hemiparesis is sometimes present in a case of temporal abscess, and the symptom may be misleading. The loss of power which is usually on the opposite side of the body is due to pressure exerted from the temporal to the frontoparietal region across the Sylvian fossa. When the abscess is of the diffuse subdural variety, the pus may invade the Sylvian fossa and exert its influence directly upon the central and third frontal convolutions, as well as upon the temporal lobe. True motor aphasia, as well as paresis or paralysis, may be then present. A more or less circumscribed abscess of the

superior temporal region of the left side gives partial or complete word deafness and paraphasia, and in some instances verbal amnesia. Paralysis of the third nerve on the same side as the abscess, due to pressure, is another not infrequent association symptom, and occasionally the fifth and sixth nerves also suffer. When the abscess is in the occipital lobe visual symptoms will be caused, and similarly in other cortical and subcortical positions the symptoms will be those of irritation or destruction, or such as have been described in detail under localization and phenomena the result of pressure and invasion of adjoining districts. Prefrontal abscesses are hard to locate by special symptoms. Frontal headache and an indifferent or apathetic mental state, and the peculiar psychical phenomena such as have been detailed both under localization and under brain tumors, are significant, and, according to Paget, great thirst and voracious appetite are often present. Extension backward of a prefrontal abscess, when on the left side, causes motor aphasia or agraphia, while in other rare instances abscesses are circumscribed in the region of Broca.

Symptoms of Cerebellar Abscess. An abscess of the cerebellum is usually situated in one of its lateral lobes, and may give rise to focal symptoms due to pressure upon the bulbar region, as well as to true cerebellar symptoms, such as ataxia and nystagmus. Rigidity of the neck and occipital headache are comparatively common, and vomiting is frequent. Facial paralysis, deafness, ear pains, and mastoid swelling may be present in cases due to aural disease. In twenty-three cases of cerebellar abscess collected by Deanesly the summarized symptomatology was as follows. Optic neuritis was definitely stated to have been absent in three out of twenty-three cases, and in eight the point was not recorded. Headache was present in all; vomiting was present in fourteen, absent in three, and not mentioned in the remaining cases. In nearly all the cases the temperature was raised above the normal in the early stages, but in all, with one exception, it sank to normal or subnormal before operation or death. Other symptoms sometimes present were slowing of the respiration, Cheyne-Stokes breathing, repeated yawnings, slowness of cerebration and general apathy, irritability and intolerance of light, delirium, rigidity of the neck and retraction of the head, nystagmus, giddiness, unsteadiness in standing, and a staggering gait with a tendency to fall laterally or forward. Excluding paralysis or irritation of the facial muscles on the same side as the ear disease, in only seven cases was motor or sensory paralysis observed. In advanced cases of cerebellar abscess, great depression and prostration, with extremely slow pulse and respiration and low temperature, are common symptoms. Unilateral paralysis due to pressure on the pons or oblongata may be present upon the same or upon the opposite side, according as the pressure is exerted above or below the

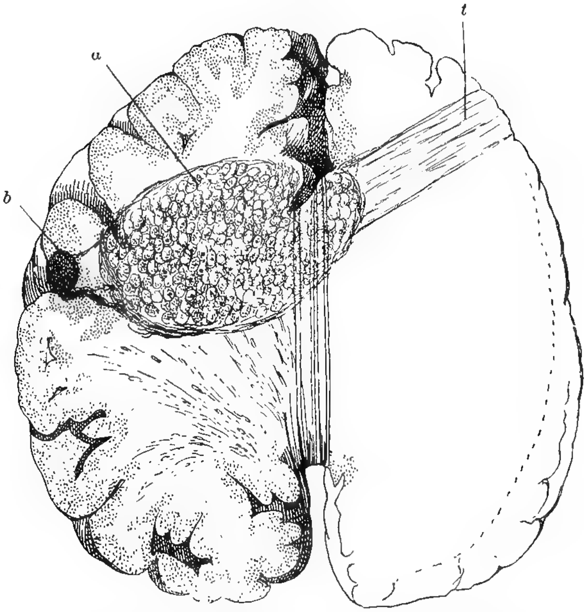
decussation. In rare instances bilateral paralysis or paresis due to pressure has been observed.

Symptoms of Diffuse Abscesses in the Cranial Fossæ. Diffuse abscesses sometimes occupy the various cranial fossæ between the dura and the brain. The pus may accumulate in these spaces as the result of disease of the bone or membranes; or it may be derived from abscesses in the substance of the brain but near its surface, and the symptoms will differ according as the abscess originates in one or the other of these ways. When the brain substance is directly involved the symptoms will be those already described as belonging to abscesses of the cerebellum, temporal lobe, or prefrontal lobe, with the addition of others indicative of diffuse basal involvement. A diffuse abscess of the anterior cranial fossa, for instance, may give disorders of smell and of the nasal branch of the fifth nerve. Pus may be so diffused in and beyond the middle fossa as to involve the fourth, fifth, sixth, and seventh nerves, or any one of them, while diffuse abscess of the posterior cranial fossa may give cerebellar and other symptoms referable to the implication of the nerves derived from the pons and oblongata, or of other structures in these portions of the brain. As the pus accumulates in one cranial fossa, it may overflow into other fossæ of the same side, into the midregions of the base, and even into the fossæ on the opposite side of the skull; so that the symptoms may show widespread implication of nerves and other structures at the base, and these may in some instances be irregularly bilateral.

Etiology.—Abscess of the brain is much more frequent in males than in females, some statistics showing a proportion of nearly two to one in favor of the former. It is most frequent between seventeen and thirty years. In children between the ages of three and ten it is infrequent, while in those under three years of age it occurs moderately often because of the tendency to middle ear disease, the thinness of the cranial bones, and the difficulty of treating aural disease in infants. From one third to one half of the whole number of cases of intracranial abscess can be traced to aural disease, and especially to suppurative affections of the middle ear. Barker and von Bergmann consider fifty per cent. to be due to otorrhea; and yet only a small percentage of the miscellaneous affections of the ear result in brain abscess. Jansen found only seven cases of cerebral abscess clearly traceable to disease of the ear in thirteen thousand cases of aural disease of all sorts. Nearly all cases of temporal and cerebellar abscess are associated with aural disease. Brain abscess is more frequent in association with chronic than with acute ear disease. A brain abscess seems sometimes to result from tubercular disease of the ear and the temporal bones, but generally when tuberculous disease is present the infection is mixed. Traumatism in the etiology of brain abscess ranks next to aural disease. While fracture is usu-

ally present, abscesses are occasionally observed after injuries which have not resulted in fracture. Traumatic abscesses are most frequent in the frontal and parietal regions, especially in the former. When the result of a gunshot wound, suppuration may follow the entire track of the ball, or the abscess may form only where the ball has lodged. The abscess may be more or less remote from the wound of entrance; or different collections of pus may be present in one or both hemispheres. In Fig. 300 is shown an encapsulated abscess in the track of a ball in a case reported by Porter. This track extended through the right and left hemispheres. The abscess was

Fig. 300.



Transverse section of an abscess, and track of the ball: *a* represents a section through the abscess with the contents *in situ*; *b* shows the ball encapsulated just underneath the pia at the bottom of the bullet track; *t* shows the cicatrized wall of the open track of the bullet from the point of entrance down to the abscess. (Porter.)

chiefly in the left hemisphere, but reached above and in front of the callosum to the right. The track of the ball in the right hemisphere had healed. In one case which fell under my observation a circumscribed abscess in the prefrontal lobe resulted from a wound in the brain by the wire rib of an umbrella, which had penetrated the floor of the skull by way of the nose. The healed track of the wire could be traced, the abscess being at its termination. Erysipelas or cellulitis of the face and scalp, and suppurative processes anywhere about the head, as in the nose or the nasopharynx, in the orbits, and along the sinuses, may be the source of the abscess. It may occur also as the result of suppurative disease situated anywhere in the body,—

from ulcerative endocarditis or endarteritis, or from purulent disease of the lungs, or even from similar disease in remote localities, as in the abdominal cavity, the pelvis, or the limbs.

Pathogenesis.—All abscesses of the brain are formed secondarily to a primary focus of infectious disease located elsewhere, the chief infectious foci being found in connection with middle ear disease. The microorganisms which give rise to abscess of the brain, as to other purulent intracranial affections, are chiefly the streptococcus pyogenes, the staphylococcus pyogenes aureus, the intracellular diplococcus, and the pneumococcus (see page 287). The parasitic fungus, *oidium albicans*, has been found in a few cases. In two cases of thrush recorded by Zenker many small abscesses were scattered through the brain, and the oidium albicans was found in the blood-vessels. In very rare instances the disease seems to have originated in actinomyces.

Pathological Anatomy.—Encephalic abscesses are usually in the white matter of the brain, or between the bone and the dura. In chronic cases suppuration is very commonly diffuse, while in the acute cases it is more liable to follow the sinuses. Abscesses associated with disease of the middle ear are more frequently on the right side than on the left. An unusually located abscess was observed in the Philadelphia Hospital wards of Dr. Sinkler and the writer, involving the internal capsule, lenticula, and thalamus, and extending forward slightly into the white matter of the prefrontal region. In cases of chronic abscess the pus is usually green and fetid, and a limiting membrane is commonly present. The greenish yellow color is probably dependent upon the bacillus pyocyaneus. Occasionally the pus is brownish or brownish red in appearance, this tint being due to hemorrhagic extravasations. In recent cases the pus is generally yellow. Brain abscesses are usually surrounded by a zone of broken down cerebral substance. The pulpy environing zone is often in a more or less inflammatory condition, and sometimes the walls of the abscess cavity are jagged and irregular and show shreds of sloughing tissue. In other cases the abscess is circumscribed and demarcated by a firm membrane. To this encapsulating pyogenic membrane the term *pyophylactic* has been applied. It is composed of condensed inflammatory tissue which in some cases forms as the active suppurative stage subsides. It may become firmer and thicker as time progresses, and it is owing to the protection afforded by it that abscesses so often remain stationary and more or less latent. Occasionally something like absorption may occur, the pyophylactic membrane becoming vascularized, and phagocytic action tending to dispose of the contents of the abscess. In other cases vigorous granulation tissue displaces the membrane, and the abscess augments in volume from within outward until it bursts and gives rise to severe acute symptoms, the case soon terminating fatally. (Park.)

Diagnosis.—Intracranial abscess may be confounded with meningitis, secondary sinus thrombosis, tumor, and occasionally with hemorrhage, with encephalitis without abscess, and with thrombotic softening. It may also be necessary to distinguish it from disease of the ear and mastoid without intracranial complications.

Meningitis. In the diagnosis of intracranial abscess from meningitis the cause and course of the two affections must be kept prominently in view. Abscess and meningitis may be due to the same causes, as to ear disease and injury. The two are often associated, in which case the existence of meningitis may be recognized and the abscess overlooked, but the latter can sometimes be diagnosticated if focal symptoms are present and receive attention. Its acute and rapid course in some cases, and in others its explosional manifestations when the disease has been for a time latent, will assist in diagnosticating an abscess from meningitis. Meningitis more frequently than abscess and also in a more irregular manner involves the intracranial nerves. The symptoms of abscess are oftener unilateral than are those of meningitis, and in this connection the existence in cases of abscess of exaggerated tendon and muscle phenomena on one side may be of value. While meningitis and abscess often have the same origin, the former has a wider range of causation. It may originate from tuberculosis or syphilis, or from any of the infectious diseases. On the whole, the presence of symptoms indicating suppuration and focal disease is most serviceable in reaching a conclusion. When pulse and temperature are persistently subnormal, an abscess should be suspected. All destructive symptoms are sometimes absent in a case of prefrontal abscess, which, however, may be suggested by the history of a traumatism or by the existence of disease of the frontal sinuses.

Sinus Thrombosis. Uncomplicated sinus thrombosis is diagnosticated from encephalic abscess chiefly by the local painful areas and edemas, and by paralyses of cranial nerves and other focal symptoms which point to closure of the special sinuses, as the lateral, petrosal, and cavernous. These symptoms have been discussed sufficiently to make the diagnosis clear, under sinus thrombosis. Attention has been called in the present section, under symptomatology, to the clinical manifestations of coexisting abscess and sinus thrombosis. When meningitis, abscess, and sinus thrombosis are all present at the same time, the confusion is increased, but even then the diagnosis can be made by close scrutiny of the external evidences of thrombosis, of the focal symptoms of the abscess, and of the symptoms of meningocortical irritation. The symptoms of thrombosis become the most prominent. General septicemia is usually present. A reddish brown discharge is regarded as diagnostic of suppurative thrombosis of the lateral sinus.

Tumor. In the diagnosis of intracranial abscess from tumor the

history is of most value. Aural disease points to abscess, and traumatism is a more frequent direct cause than of tumor. The symptoms are more regular and uniformly progressive in tumor, and when spasm is present signal symptoms are not so common as in the latter. These and other points of distinction are summarized in the following tabular statement of the differential diagnosis of brain tumor and abscess:

Abscess.	Tumor.
Suppurative disease in the ear or elsewhere common.	Suppurative disease usually absent.
History of traumatism frequent.	History of traumatism not uncommon, but not so frequent.
Onset of active symptoms either abrupt or slow.	Onset usually slow.
Headache usually dull.	Headache of great intensity.
Papillitis may be present, but oftener absent.	Papillitis more commonly present and usually of higher grade.
Febrile symptoms often those of pyemic infection.	Febrile symptoms not those of purulent disease.
Rigors and sweating frequent.	Rigors and sweating usually absent.
Temperature and pulse medium or subnormal.	Temperature and pulse variable.
Diffuse meningitis often associated.	Meningitis less common, and usually localized around the growth.
Phlebitis and sinus thrombosis frequent accompaniments.	Phlebitis and sinus thrombosis usually absent.
When spasm occurs, signal symptoms not common.	Signal symptoms frequent.
Focal symptoms most frequently indicate the temporal lobe or the cerebellum.	Focal symptoms most frequently motor, but may indicate any part of the brain.

Encephalic Hemorrhage. Occasionally the diagnosis between abscess and encephalic hemorrhage may present difficulties, as when an abscess which has been apparently quiescent suddenly ruptures and gives rise to symptoms of an acute apoplectic attack. The presence of suppurative disease in the ear or elsewhere, the occurrence of rigors, and the conditions of temperature and pulse indicative of purulent affections will here be of value. As abscesses do not usually occupy the position in which hemorrhages are found, motor symptoms are often not prominent; but, as already stated under focal symptoms, paresis or paralysis may be due to the pressure exerted by a temporal abscess. An abscess which bursts into the ventricles from any quarter may give symptoms like those of secondary ventricular hemorrhage. In one of my cases referred to under pathological anatomy, in which an abscess was found occupying the usual seat of intracranial hemorrhage, the patient, a woman, twenty-five years old, had purulent disease of the left ear. She complained of pain in the right side of the head and neck. Her temperature was

102.4° F., and two days later it rose to 103° F., and she soon passed into a stuporous state. She had mastoid tenderness upon the right side, with edema of the neck in the vicinity. Later a purulent discharge took place in the left ear. The right pupil was contracted, and both pupils reacted to light. The left face, arm, and leg were paretic at first, and later the limbs showed well marked paralysis with spasticity. In both leg and arm at one stage were scattered areas of partial or delayed sensation which in some places amounted to complete anesthesia. The knee and muscle phenomena and cutaneous reflexes were exaggerated on the left side. Edema of both optic disks was present. The patient was irritable, restless, at times delirious, and at others stuporous. She showed considerable variations in her general condition during the three months preceding her death; but the hemiplegia persisted. Pulse and temperature were sometimes subnormal. The symptom picture in this case was confusing, but the history was not that of a sudden attack. This case is recorded by Dr. E. A. Shumway in Vol. III. of the Philadelphia Hospital Reports. Acute softening from either embolism or thrombosis might in rare instances be confounded with abscess of the brain, but the diagnosis can be made by a consideration of the etiology, by the presence of symptoms indicating suppuration, and by a study of the points given in the table of the differential diagnosis of hemorrhage, embolism, and thrombosis on page 489.

Encephalitis. Acute nonsuppurative encephalitis, either focal or diffuse, is wanting in the symptoms of septic infection, and runs a definite and often rapidly progressive course without intervals of latency or partial latency. Focal encephalitis is more frequently than abscess an affection of the motor regions of the brain. Diffuse subcortical encephalitis has mental vacuity or torpor as a more persistent and distinctive symptom. Diffuse suppurative encephalitis, without circumscribed accumulations of pus, as distinguished from abscess as a focal disease, is briefly considered on page 542.

Suppurative Aural Disease, without Abscess. The local or peripheral irritation caused by a diseased ear may give rise to symptoms which closely resemble those of intracranial abscess. Dizziness, giving with tinnitus and deafness Ménière's symptom complex, may be present, as also headache and vomiting. In such cases the physician should carefully weigh all the manifestations in the case, laying particular stress upon papillitis, aphasia, ataxia, hebetude, delirium, or other symptoms, especially focal manifestations of intracranial disease.

Prognosis.—The prognosis is always grave. The acute cases, while usually rapidly fatal, are the most amenable to treatment. Occasionally striking successes are obtained by surgical interference, and much attention has been paid to this subject by neurologists and surgeons in recent years. Recovery is more frequent from temporal than from other forms of encephalic abscess. In a very few cases

abscesses of small size become firmly encapsulated, and partially disappear by absorption or by phagocytosis, the patient dying of some other affection. In exceedingly rare cases abscesses have evacuated themselves spontaneously. In fatal cases death results in various ways. It may in some cases be due as much to a widespread meningitis or to sinus thrombosis as to the abscess. The abscess may rupture into the ventricles, leading to a speedy fatal issue; the centres of the bulb may become involved; or the end may come through general pyemia or exhaustion.

Treatment.—*Medical Treatment.* In every case of purulent disease of the ear—indeed, in every case of chronic aural disease with a recurring tendency to suppuration—the possibility of intracranial abscess should be borne in mind, careful attention to such affections constituting one of the most important prophylactic measures against abscess of the brain. The general health should be sustained. Tonics, nutrients, fresh air, and the avoidance of exposure and injury should form a part of the management of the case. For acute symptoms the treatment is practically the same as that for meningitis or encephalitis, or for any irritative disease of the brain. It includes counterirritation, local depletion, the application of cold or heat to the head, and the use of remedies to subdue the fever, control pain, and counteract septic infection.

Surgical Treatment. Like cranial fractures and intracranial hemorrhage, abscess of the brain often calls for prompt trephining. It is an interesting historical fact that in 1871 Paul Broca, through speech disturbances, localized a cerebral abscess over the left third frontal convolution, and on trephining found pus between the bone and the dura. The patient subsequently died of meningoencephalitis, a fatal issue that would probably not occur in these days of aseptic and antiseptic surgery. Barker makes a broad assertion, which is nearly true, that nine tenths of all encephalic abscesses are situated within a circle three fourths of an inch in diameter, the centre of which is an inch and a half above and behind the centre of the meatus. The most important points for trephining are given in the section on craniocerebral topography. If the presence of an abscess is determined and it is accessible, it should be operated upon as speedily as possible. When the symptoms indicate the locality of the lesion, the duty of the surgeon is to operate guided by them; if they are more general and unrelieved by medical treatment, an exploratory opening should be made at some point where no important intracranial vessel or sinus is likely to be injured, and if pus is obtained a second or counter opening should be made. It is important to remember cases such as that recorded by Drummond which indicate how erratic and anomalous the symptom picture of encephalic abscess may be, and also how a mistake in operating may occur. The patient, a girl nine years old, had for several years a purulent discharge from the right

ear. Headache, vomiting, right-sided convulsions, and paralysis of the right arm gradually passing into right hemiplegia, rather suddenly developed. Later she was aphemic and optic neuritis was present. Cotton introduced into the right meatus was quickly moistened with a purulent fluid, although there was no active discharge on either side. A trephine opening without result was made over the left temporal lobe. A second opening was made over the arm centre, and a third over the left lobe of the cerebellum, but with equally negative results. The autopsy revealed nothing in the left hemisphere except the evidences of the operation, but an abscess cavity holding about an ounce of pus was found in the right lobe of the cerebellum near the surface.

MULTIPLE ENCEPHALIC ABSCESS.

The possibility of multiple abscess is important from both medical and surgical points of view. General pyemia or septicemia may give rise to multiple abscesses, one or more of which may be within the cranial cavity, and even from a single source of infection a number of abscesses may originate. Finley and Adami have reported a case in which half a dozen pus cavities were found in the white and gray matter of the brain, the source of infection having been a suppurating bronchial gland secondary to pneumonia, a not infrequent source of cerebral abscess. Nauwerck has reported another case with depression and perforation of the right temporal bone, defect of the underlying convolutions, and multiple abscess and hydrocephalic softening of the hemisphere. Corresponding to the traumatic depression of the bone, eight distinct abscesses were found filled with thick green pus. Twenty-eight years elapsed between the reception of the injury and the final fatal result, probably the longest recorded time of quiescence in cases of traumatic intracranial abscess. Eskridge and Parkhill have reported a case in which two abscesses were present in the same hemisphere, one of which was successfully opened and the other was overlooked. Both were encapsulated, and no connection existed between them. Multiple abscesses are very common in the same half of the brain, or they may be multiple in the cerebrum, the cerebellum escaping, or the reverse. It may be difficult or impossible to determine that a second or a third abscess is present in the brain after one has been evacuated, and especially when cerebral symptoms are complicated by those of the primary morbid condition in the ear, lungs, or elsewhere. Occasionally fresh abscesses form in the neighborhood of one that is encapsulated, the tissues being in an unhealthy condition and liable to inflammation. A new abscess or new abscesses may result from infection due to leakage from the original purulent focus. A case is described by Macewen in which an extradural abscess formed so completely around the old one that the capsule containing pus was found floating in the secondary abscess.

CHAPTER VI.

RESIDUAL ENCEPHALIC LESIONS, DEGENERATIONS, AND DISEASES.

ACUTE focal diseases of the brain, such as hemorrhage, softening, tumor, and abscess, when they do not result fatally, leave cystic, necrosed, or sclerosed areas, and these lead to progressive degenerations of the central and peripheral nervous systems. Special symptoms and syndromes are also developed, and these, although they preserve from the first some of their characteristic features, are modified in accordance with the progressive pathological changes. To some of these changes attention has been already directed, as when reference was made to the consecutive alterations in a clot after hemorrhage into the brain. Allusion has also been made to secondary degenerations, but these and their concomitant clinical phenomena—the hemiplegias, monoplegias, anesthesias, spastic disorders, aphasias, hemianopsias, and other residual affections—require further and special consideration. These conditions often persist for years, and it is with them that the physician frequently has to deal, rather than with the primary and causal affections.

SECONDARY DEGENERATIONS CHIEFLY AS OCCURRING AFTER ACUTE FOCAL LESIONS IN ADULTS.

Cruveilhier in 1832 observed diminution in size of the pyramidal tract in a case of hemiplegia. Türck, from 1851 to 1855, first systematically described the secondary spinal degenerations following cerebral lesions; since that period, and especially during the last ten years, the subject has been much advanced by physiological and histological researches. One of the most important clinico-histological contributions to the subject is Tooth's Gulstonian Lectures on Secondary Degeneration of the Spinal Cord, published in 1889. While investigations with the Golgi and Marchi methods of staining are changing in detail the former views with reference to secondary degenerations, some of the most important theories regarding degeneration which follows focal cerebral lesions remain as advanced by Türck, Charcot, and Bouchard. The distinctive bands of secondary degeneration caused by the acute focal diseases of the brain can be readily isolated and studied. The diffuse affections of the encephalon, as chronic meningitis, encephalitis, and even endarteritis, also lead to secondary changes, but these are of an unsystematic character. Such degenerations result when meningitis and lesions of the surface of the brain cause cortical destruction deep enough to involve the ambiguous and great pyramidal layers. Tumors do not give rise to secondary de-

generations as the result of pressure, but after they have produced some destruction of brain substance. Secondary degeneration is set up and progresses chiefly in the conducting tracts along the lines in which they transmit motor, sensory, or other impulses. In the motor system the degeneration is descending or centrifugal—from the forebrain towards the pons and oblongata, from these structures towards the spinal cord, and from the spinal cord towards the periphery. In the sensory systems it is ascending or centripetal —from the peripheral sense organs to the dorsal ganglia or cord, and from the cord to the brain; from the organs of special sense in the primary basal centres, and from these and their tracts to the higher regions of the cerebrum.

Initiation and Progress of Secondary Degeneration.—Probably as soon as a nerve fibre is totally separated from its trophic centre it begins to degenerate; but the changes which take place are not appreciable for several days. Homén placed the earliest time at which such alterations could be recognized at about the third day. Tooth found evidences of degeneration in the spinal cords of lower animals and of man after six days; and the presence of secondary degeneration cannot be affirmed by a study of clinical phenomena or by microscopical appearances until about this time. Boyce, whose investigations were made upon the hemicerebrums of cats by Marchi's osmic acid method, discovered traces of degeneration five days after the excision of the hemispheres. Histological investigations would seem to show that secondary degenerations cannot be appreciated quite as early in the adult human being as in the lower animals. Just when the process of secondary degeneration in a given tract is completed it is difficult to say. It certainly continues for six months or more, and may in some instances not be completed for one or two years. Different tracts probably have different rates of progressive degeneration.

Inflammatory Degeneration.—It is equally important to the histologist and to the clinician to remember that near the initial lesion, especially if it is irritative in character, these changes, which are partly degenerative, must be distinguished from those of secondary degeneration proper. Under the name of *traumatic degeneration*, these changes have been particularly studied by Schiefferdecker, Homén, and Tooth. This inflamed and degenerated tissue is found both above and below the lesion, for the distance of one centimetre or more. Doubtless some of the peculiarities in the early symptomatology of cases of hemiplegia are due to the presence of this tissue. While the studies of the changes present near the initial lesion have been chiefly made upon traumatic spinal cases, the facts determined are equally applicable when encephalic lesions are of an irritative character, such as tumors, traumatisms, hemorrhages, abscesses, and encephalitis.

Distinction between True Secondary Degeneration and Involution.—Secondary degeneration is commonly due to the separation of the nerve fibres or processes from their trophic centres, the cell bodies. A distinction must be made between true secondary degeneration and involution. The former extends only to the extreme limits of the fibres which are cut off from their trophic centres. When, for example, a destructive lesion is in the cerebral cortex, centrum ovale, or internal capsule, true secondary degeneration can be traced throughout the pyramidal tract to the nuclei of the cranial motor nerves in the bulb, and to the nuclei of the spinal motor nerves in the ventral horns of the cord, but not into these gray masses themselves nor into their distal processes. The same is true of ascending secondary degeneration, which extends from a peripheral lesion to the dorsal ganglia, or from a lesion in the cord to the gray masses interposed in the sensory tracts at the lower extremity of the oblongata. The process of disappearance or partial disappearance of intraspinal and intraencephalic centres and tracts, known as involution, is somewhat different. Involution is a retrogression which certain structures undergo as the result of disuse. The tracts and centres in this process atrophy because they have ceased to function, not because the trophic centres from which their fibres originate have been cut off or destroyed. After evulsion or destructive lesions of the peripheral nerves, the parts related to these nerves, both in the spinal cord and in the brain, undergo more or less involution. After amputation of a limb, areas in the opposite cerebral hemisphere, particularly in the motor region, have been found greatly atrophied. After removal of the eyeball, or after long continued blindness, related central parts as far as the occipital lobe undergo involution. In like manner, after destruction of the aural apparatus or after long continued deafness, a similar involution is noted in the encephalic auditory tracts and centres as far as the temporal cortex. Involutions of the cerebral visual area have been described by me as occurring in the brain of a woman who was blind for more than twenty-five years, the occipital lobes having been very small, and of the cerebral auditory areas in the brain of a man deaf for thirty years, the supertemporal convolution having been unusually small and smooth. The brain of the blind deaf mute Laura Bridgman, thoroughly investigated by Donaldson, showed involutions of portions of the occipital and temporal lobes and other regions of the brain. Ascending secondary degenerations from transverse lesions of the spinal cord extend as far as the clavate and cuneate nuclei, but when such cases are of long standing, involution takes place in the sensory centres and tracts, basal and cerebral, above these nuclei. Motor cerebral areas because of disuse undergo involution after diseases such as anterior poliomyelitis, which destroy spinal centres and lead to neural degeneration and paralysis. A striking

illustration of involution is that of an entire lateral lobe of the cerebellum after a large destructive lesion or arrest of development of the opposite hemicerebrum. Destruction of one occipital lobe leads to involution not only of the optic radiations, pregeniculum, pregeminum, and optic tracts, but also of special association tracts connecting this lobe with the parietal and other regions of the cortex. Excision or destruction by disease of the superior temporal convolutions is, in like manner, followed by degeneration and involution of the intracerebral centres and tracts concerned with hearing and speech.

Secondary Degeneration of the Crossed Pyramidal Tracts.—The great pyramidal tract is the seat of the most important form of secondary degeneration following encephalic lesions. A destructive focal lesion of any portion of the motor cortex or subcortex causes degeneration in this tract below the focus of disease, which degeneration is greater or less according to the extent and destructiveness of the originating lesion. The ganglion cells of the central or Rolandic region are the chief trophic centres for the fibres of the upper levels of this great tract. Türck believed that degeneration of this tract also originated from destruction of the basal ganglia, and especially of the striatum, and it may be that the tract is fed by some fibres from the basal ganglia. As far caudad as the pyramidal decussation, secondary degeneration following unilateral lesions of the motor portion of the cerebrum is confined to one pyramidal tract, that of the side of the lesion. It has been long known that below the decussation the degeneration continues through the entire length of the lateral columns of the spinal cord, on the side opposite to that of the lesion in the brain, the so-called crossed pyramidal tract. It has also long been known that descending degeneration of the direct pyramidal fasciculus in the anterior column takes place; but this is of limited extent. More recently it has been demonstrated that degeneration of the lateral pyramidal tract on both sides is present, and usually of a marked character. These bilateral degenerations will be next considered.

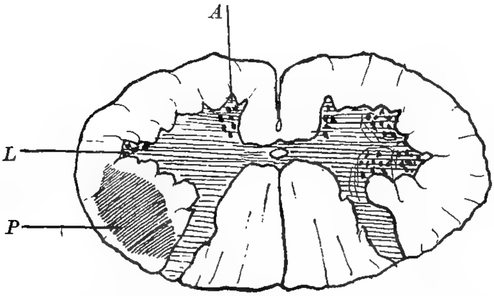
Secondary Bilateral Degeneration of Motor Tracts.—Charcot early observed bilateral degeneration, and it has since claimed the attention of many others. Pitres in 1884 found that of ten cases, in six the bilateral degeneration was symmetrical in the pyramidal bundles of the lateral columns of both sides, while in four it was greater on the side opposite the lesion. Türck's direct fasciculus in the anterior column, in two of the four cases in which the pyramidal degeneration was unequal, was normal on both sides. Sherrington, in 1885, from a series of experiments on the cortex of the dog, concluded that injury of the "cord area" (motor area) of one hemicerebrum caused degeneration in both halves of the spinal cord in the dorsal angle of the lateral columns, and held that the anatomical

changes were the results of the clinical symptoms becoming bilateral. He also showed that bilateral degeneration takes place in the pons and oblongata after a unilateral focal lesion. Many advances have been made upon these investigations, and even on those of later date. The researches of Muratoff, Boyce, and Mellus place beyond doubt the fact that the fibres of each pyramid split into two sets. One of these, the larger of the two, goes to the lateral tract of the opposite side, the other to the corresponding tract on the same side. As the result of some experiments on cortical centres it was found that bundles of degenerated fibres on the side of the lesion were present in amounts varying from one tenth to one third of those in the crossed tracts, the conclusion being that the bilateral degeneration observed by previous investigators indicates a general bilateral descent of fibres from one hemisphere. A few degenerated fibres were found in the direct tracts of the anterior columns. The pyramidal tracts in the cord, it will be seen, are therefore composed in part of fibres of heteromeral and in part of those of hecatomeral cells (see pages 92 and 93). Secondary degenerations of the motor tract sometimes show great peculiarities and striking departures from the ordinary rules. Most of them are best accounted for by congenital absence, deficiencies, or aberrations of this tract, or by fetal lesions, or by affections occurring at or near the time of birth. Sometimes the degeneration is irregular, and in rare instances no crossing takes place. In some cases of hydrocephalus the crossed pyramidal tracts have been found entirely wanting; but in these instances they may have been congenitally absent.

Degeneration following Encephalic Lesions variously located.—Various more or less isolated tracts of degeneration besides those so long known in their entirety in the pyramidal bundles have been traced in the lower animals and in man by a few observers, but as yet not much light has been thrown upon the functions of these tracts. Sherrington found that, in addition to the pyramidal tract degeneration, other regions of the pons, oblongata, and cord degenerated or involuted after unilateral encephalic lesions, as the central gray and ventral horns of the cord; the lateral horns of the cord; islanded gray masses in the pons lying close to the fibre bundles of the crustal tract in the deep transverse fibres; a gray mass in the mesal third of the crusta; and the intercalatum or substantia nigra, especially its ventral portion. In Fig. 301 is shown the relative preservation of the anterolateral gray matter of the cord on the two sides in a case of unilateral lesion of the brain resulting in hemiplegia with some atrophy, crossed pyramidal degeneration being also shown. Such a case is in accord with some of Sherrington's experiments on lower animals. In a case recorded by Bechterew, the substantia nigra was markedly degenerated as the result of a lesion which involved the cortex, centrum ovale, capsules, and ganglia. This degeneration was,

however, part of a general secondary process which caused a shrinkage of the entire pes and the same side of the pons. In the oblongata

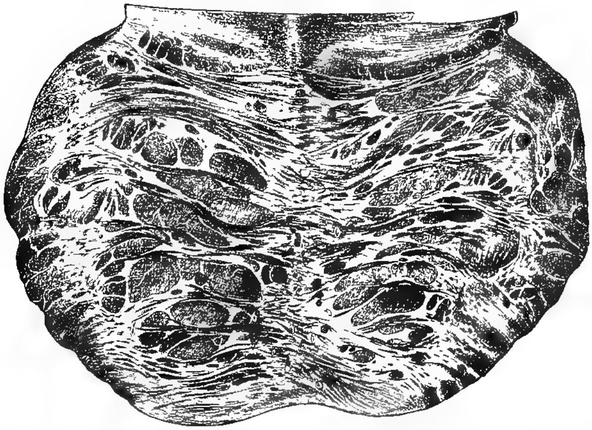
FIG. 301.



Section of the spinal cord in the cervical region in a case of left hemiplegia accompanied by amyotrophy: *A*, anterior cell groups; *L*, lateral cell groups; *P*, degenerated crossed pyramidal tract. In the anterior horn of the left side some cells of the anterior group, *A*, and of the lateral group, *L*, are alone preserved; all the others are atrophied or have undergone involution. (Marie, after Pitres.)

it was restricted chiefly to the pyramidal bundles of the same side, and in the spinal cord to the pyramidal tract of the opposite side. Bechterew attributed the degeneration of the intercalatum to the destructive lesion of the basal ganglia, and particularly of the striatum.

FIG. 302.



Section at the level of the postgeminum showing secondary degeneration of the pyramidal tract and other structures of the same side, in a case of large lesion of the thalamus and partial destruction of the posterior portion of the posterior limb of the internal capsule.

The illustrations (Figs. 302, 303, 304, 305) show the most important degenerated tracts following a large thalamic and capsular lesion in a patient who was for many years before her death under my care in the Philadelphia Hospital. She had an apoplectic attack in 1877, and died in 1892. The summarized symptomatology of the case was

hemiparesis with contractures, and hemianesthesia with inability to recognize the position of the affected limbs. Hemianopsia and all affections of the special senses were absent, as were also athetoid and choreoid movements. Autopsy showed an old hemorrhagic cyst,

FIG. 303.

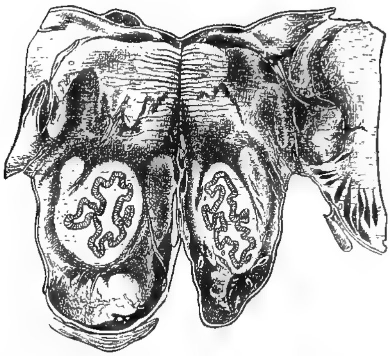


FIG. 304.

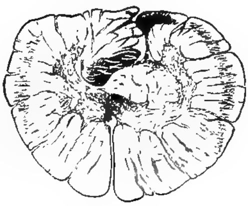


FIG. 305.

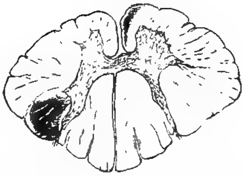


FIG. 303.—Section of the oblongata, showing secondary degeneration of the pyramidal tract, changes in the olivary body, and general shrinkage of the entire oblongata on the side of the lesion (same case as in Fig. 302).

FIG. 304.—Section through the pyramidal decussation, showing marked degeneration of the pyramidal tract of both right and left sides (same case as Fig. 302).

FIG. 305.—Section just below the decussation, showing marked degeneration of the crossed pyramidal tract of the left side, and slight degeneration of the direct pyramidal tract of the right side (same case as Fig. 302).

which had destroyed about two thirds of the substance of the thalamus, including the entire external tubercle and a large portion of the pulvinar, the anterior extremity and the internal and inferior surfaces of the thalamus being intact. The lesion had also invaded the posterior half of the posterior limb of the capsule. The sections all show marked degenerations of the great pyramidal tract. Degeneration of the central tegmental tract of the right side was also present. This tract, first described by Bechterew, has been chiefly demonstrated by developmental investigations. Appearing on the posterior and external aspect of the olive, it gradually increases in size from the middle of the olive upward. The section through the oblongata (Fig. 303) shows marked degeneration of the pyramidal bundle, changes in the inferior olive, and shrinkage of the entire oblongata on the side of the lesion. The other sections (Figs. 304, 305) show marked degenerations both at and just below the decussation.

Tracts of Degeneration in the Crus. From a study of different cortical lesions Zacher differentiated various tracts in the crus. He divided the peduncle from without inward into four nearly equal parts. The fibres which run in the outer fourth were found to come from the occipital and temporal lobes, and to reach the upper levels

of the pons, in which they probably terminate. In an instructive case of congenital atrophy of the occipital lobe recorded by Kreuser, this tract and other important structures were lacking. The second fourth of the crus from without inward was found by Zacher to be in great part occupied by the pyramidal tract, which is in accord with the observations of Flechsig and of others. In the crus it is the chief seat of the intraencephalic degeneration described in the last paragraphs. According to Zacher, the fibres of the third fourth of the pes, measuring from the external border, probably arise in part in the body of the striatum, and also in part from the ascending frontal convolution. Flechsig supposed that the median or inner fourth of the crus received its fibres from the prefrontal lobe, which is the view generally held. While degeneration of this band seems to cease in the pons, it is probable that through the intermediation of pontile cell nests it is connected with the cerebellum, and its fibres are therefore sometimes termed frontocerebellar. Secondary degeneration of this tract has also been found after disease of the most anterior portion of the internal capsule. Zacher's view of the origin of these fibres of the innermost fourth of the crus is different. According to him, they are derived from the neighborhood of the insula and base of the lenticula.

Degeneration of the Lemniscus and of Various Special Tracts. Bruce has recorded the careful microscopical study of a case in which, in addition to degeneration of the pyramidal tracts, dorsal longitudinal bundle, and other structures of the oblongata, there was marked degeneration of the lemniscus after an old and extensive lesion of the cerebrum, the parts destroyed being large portions of the internal capsule and of the basal ganglia. The cephalic two thirds of the callosum and a large area of the centrum ovale were atrophied. The nucleus cuneatus and the nucleus gracilis on the opposite side of the lesion were considerably less than the same structures on the same side. Bruce believes from his observations in this case that the nucleus cuneatus is more directly connected than the nucleus gracilis with those fibres of the fillet that end in the cerebrum, which is in harmony with the results obtained in the embryo. While the fillet has thus been shown to degenerate downward, it is known that the posterior columns do not degenerate in this direction. A unilateral destructive lesion at about the level of the postgeminum and nuclei of the third nerve was found by Boyce to cause a peculiar form of bilateral degeneration simulating closely the appearances of two degenerated pyramidal tracts. He found that these degenerated fibres near their origin cross to about the position of the fillet, and continue down the cord close to and partially intermingled with the fibres of the pyramid after their decussation. They pass down the cord as far as the upper dorsal region, lying forward and close to the middle line of the anterior column, in the position of the so-called direct pyramidal

tract. After lesions destroying either the superficial or the deep transverse fibres of the pons, secondary degenerations have been observed in the fibres on both sides of the lesion. This is what might be expected, as these fibres connect the cerebellar lobes, or one lobe or lobule of the cerebellum with the cortex or ganglia of the opposite hemicerebrum. In one of my cases a hemorrhagic cyst, which was small, but large enough to have caused destruction of numerous transverse fibres near the middle of one half of the pons, was present, and one cerebral and the opposite cerebellar hemisphere were markedly atrophied. The patient was hemiplegic for many years.

Ascending Secondary Degeneration.—The study of ascending secondary degeneration belongs in the main to the study of spinal lesions, primary and secondary. It should be glanced at here in connection with intraencephalic secondary degeneration, as some of these degenerative and involutional processes which begin at various levels of the cord do not cease until they have passed cephalad considerably beyond the foramen magnum. After reaching certain levels they cease to be true secondary degenerations, but beyond these points in some instances they are continuous to the mantle of the forebrain and cerebellum as involutions secondary to lesions and degenerations of lower levels. It has been clearly shown that after transverse lesions of the cord ascending secondary degenerations take place in at least four localities—in two triangular areas at the most posterior and mesal parts of the posterior columns, in the direct cerebellar tract, and in the anterolateral tract of Gowers. The degenerations in all these tracts become more restricted as they approach the brain. In the dorsal or posterior region the degeneration appears as a steadily narrowing band in the columns of Goll, as far as the termination of the latter in the nucleus gracilis and nucleus cuneatus. The direct cerebellar tract much condensed can be traced as far as the restis; and Tooth has traced secondary degeneration in the anterolateral tract of Gowers to the exit of the sixth and seventh cranial nerves. This column is supposed to end in the lateral nucleus.

Secondary Degenerations after Cerebellar Lesions.—In a previous section (pages 71 and 72) attention has been directed to the course of the conducting tracts of the cerebellum and of the cerebellar peduncles. After ablation of the middle lobe of the cerebellum in dogs, Pelizzi found (1) complete degeneration of the prepeduncle; (2) degeneration of the medipeduncle and of the pons, that in the pons being most considerable in the deep stratum; (3) partial degeneration of the restiform body; (4) degeneration of the fillet or lemniscus, most intense in the lateral or inferior fillet; (5) degeneration of Deiters's nucleus; (6) degeneration of the posterior longitudinal bundle; (7) degeneration of the anterior root bundles of the spinal nerves.

CHRONIC HEMIPLEGIAS AND MONOPLEGIAS—THEIR CLINICAL FEATURES, INCIDENTS, AND ACCIDENTS.

The lesions are commonly of the motor regions of the brain, and paralysis more or less complete of one side of the body is usually the result of an apoplectic attack, and according to its extent this is termed a *hemiplegia* or a *monoplegia*. As the paralysis and the conditions which accompany it at the time of an apoplectic attack and immediately afterwards have already been described, the phenomena now to be especially considered are those of the residual or chronic affection. A chronic hemiplegia is left after an acute apoplectic attack; or it is a progressive affection, which comes on step by step, as in a slowly developing meningitis or brain tumor, or when one vessel after another closes from time to time; or a progressive hemiplegia associated with aural disease may be due to a gradually forming abscess. It may be well to define here more explicitly than hitherto the meanings of the terms most commonly used in describing these paralytic affections. In the section on symptomatology and methods of investigation (see pages 165, 166) hemiplegia was defined as a form of paralysis which attacks the most of one half of the body. A *monoplegia* is a paralysis limited to one arm or one leg or to one side of the face, or, it may be, to certain groups of muscles in any one of these parts. When the monoplegia is of the face it is called *facial;* when of the arm, *brachial;* when of the leg, *crural;* and when of the tongue, *lingual;* or it may be brachiocrural, brachiofacial, faciolingual, or some other form of combined monoplegia. Monoplegias and hemiplegias are not always of encephalic origin; but our concern here is with those which are due only to brain disease. A distinction must occasionally be made between a hemiplegia and a *double monoplegia.* The entire paralysis in a hemiplegia is, as a rule, due to a single lesion, which may be large or of moderate size. In a double monoplegia the paralyses of different parts are due to distinct lesions. It is illustrated by a case in which the paralysis is in a leg, or in a leg and the face on the same side, and is due to separate lesions which have occurred at the same time or at different times; but of two attacks of cortical or subcortical embolism one might lead to paralysis of the arm or the face or of both, and the other to paralysis of the lower extremity. The *diplegias* or *double hemiplegias* which affect a large portion of both halves of the body are usually due to lesions on both sides of the brain, and constitute one variety of infantile cerebral palsies, which will presently be considered. In rare instances they may come on in adult life.

The Most Important Hemiplegic Phenomena.—Chronic "hemiplegics," as the cases just defined are usually classed, present special clinical concomitants, incidents, and accidents. The most important of these, after the paralysis, are certain disorders of move-

ment, of reflex action, of sensation; of vasomotor, secretory, and trophic functions; and of mentality. These hemiplegic phenomena are due, in the first place, to the persistence of the original focal lesions; secondly, to certain consecutive pathological changes, and especially to the secondary degenerations discussed in the last section; thirdly, to inflammatory, nutritive, or other local changes in the paralyzed parts. These last are sometimes accidents rather than true concomitants or incidents. The phenomena of secondary degeneration develop step by step during weeks, months, and perhaps years after the apoplectic seizure.

Amount and Order of Recovery in Paralyzed Parts.—Occasionally a case of hemiplegia remains for months and years almost totally paralyzed in the extremities, and with a large degree of loss of power in the lower face, no restoration of power taking place except during the first few weeks after the apoplectic seizure; but such cases are exceptional, some improvement usually showing itself as time progresses, this in some instances becoming very considerable. As a rule, improvement in the lower extremity precedes that in the upper, and the proximal portions of the limbs regain power sooner and to a much larger extent than the distal. While the tendency is for the leg to recover some power sooner, and eventually to recover to a larger extent, than the arm, occasionally cases are seen in which the latter recovers more rapidly than the former. This was the order of events in seven out of two hundred cases observed by Bastian. Trousseau taught that cases in which the arm tended to recover before the leg were usually of ill omen, and this view has crept extensively into neurological literature. He believed that dementia and a fatal issue were much more likely to occur in these cases. Like Bastian, I have seen some cases in which recovery in the arm has taken place sooner and to a greater extent than in the leg. Bastian inclines to the view that in these cases the lesion is likely to be below the capsule, and especially in the pons. While this may be true, a small lesion of the centrum ovale or of the internal capsule might be so situated as to cause destruction only of the projection fibres related to the lower extremity, such a lesion at first affecting by pressure the arm and the face fibres, the paralysis in these parts passing away as the pressure disappears. Movements of the shoulder and upper arm and those of the muscles of the pelvis and thigh are regained earlier and more largely than those of the forearm and hand or of the leg and foot. The exceptions to this rule are few. The reasons for this order of recovery are to be sought for not so much in the site and ramifications of the persisting lesions as in the part played by the undamaged hemisphere. Movements of both shoulders and of the hip and thigh—proximal limb movements generally—are represented in each hemisphere to a greater extent than are distal movements, so that one hemicerebrum learns to act for both sides,

as it does under normal conditions to a larger degree for the trunkal, laryngeal, and other movements which have an almost equal bilateral representation.

Relative Involvement of Different Parts.—As already intimated, the term hemiplegia is not strictly correct in an etymological sense. The paralysis is one-sided, but does not implicate an entire half of the body. As a rule, the muscles of the trunk and abdomen, the masticatory and the laryngeal muscles, and in general those of both sides of the body which habitually act together, escape in whole or in large part. Some of the reasons for this have been already indicated under cerebral localization (pp. 339, 340). Muscles which act synchronously, and which have a bilateral representation in each cerebrum, are completely paralyzed only as the result of bilateral lesions. Usually, therefore, a hemiplegia is chiefly of the arm and leg, and of those muscles of the face which are in the main unilaterally innervated by cortical centres. The bilaterally innervated muscles, however, do not always escape. In early infancy the unilateral differentiation of cortical centres is comparatively slight. In one of my cases, an infant who died of cerebral hemorrhage, the muscles of the trunk and face were almost equally paralyzed with those of the limbs. Even in adults, particularly in some of the severe types of hemiplegia, close inspection, especially during the apoplectic period, will sometimes show diminished power in the trunkal muscles of the paralyzed side. This may be observed in movements of respiration. No respiratory movement, or only a slight one, is seen on the paralyzed side of the chest even on forced inspiration. Broadbent was the first to suggest a satisfactory explanation of the mechanism of this retention of power, relatively or absolutely, in certain groups of muscles on the paralyzed side. He believed that the structural connections which allowed both sides to be excited from one hemisphere were between the lower centres, bulbar and spinal. According to this hypothesis, impulses coming from one side of the brain are conveyed to the lower centres on the opposite side, and the impulses designed for muscles which act habitually together are transmitted by well worn and easily traversed pathways to the homologous centres of the opposite side. Such an explanation, while sufficient for some of the phenomena, will not answer for all. Bearing upon this question is the fact that a large amount of degeneration takes place in the lateral columns of both sides after unilateral cerebral lesions, showing that each hemisphere is directly related by special fibres with the oblongatal and spinal centres on both sides. Sherrington advanced the view that some of the fibres after crossing at the pyramidal decussation recross at lower levels of the cord; but experiments show that the pyramidal fibres split into two sets, one for each side. This subject has been already discussed under secondary degenerations of the motor tracts (pages 559 and 560).

Clinical Types of Hemiplegia.—Numerous clinical types of hemiplegia can be described in accordance with the degree and special characteristics of the paralysis. As has been stated, the hemiplegias arrange themselves into preponderating arm types or leg types, and also into distal and proximal types, the upper extremity and distal types being by far the most frequent. A few special remarks might be made here about the cases met with now and then which differ markedly from the usual types, particularly as regards the proximal and distal distribution of the paralysis, and the peculiarities of other motor disturbances present, such as spasm, contractures, and ataxic or athetoid phenomena. A case, for instance, may present a paralysis of common type in the leg, more distal than proximal, and without any unusual characteristics, while in the upper extremity the paralysis is pronounced or more marked in the shoulder or the upper arm, the movements of the forearm, hand, and fingers being relatively well preserved, but showing greater incoordination and grotesqueness. Phenomena of this kind should suggest that the lesions are not those usually present in hemiplegics. They may be cortical or subcortical growths. Inquiry will often show that convulsions of unilateral type occur at intervals in such cases. A departure from the hemiplegic type usually seen should cause the physician to direct his attention to the possibility of a lesion of unusual character; and this is one of the ways in which the study of clinical types of hemiplegia and monoplegia may be of considerable practical value. Clinical types of hemiplegia which are due to the association of the paralysis with other important manifestations are somewhat frequently observed. Partial or complete anesthesia may be present. When anesthesia is present the motor paralysis may preponderate in the lower extremity or it may be associated with nearly equal paralysis of both leg and arm, the face escaping in whole or in part. Partial or complete aphasia of sensory, motor, or sensorimotor form may be combined with the paralysis. In still other cases the association may be of hemiparesis or hemiparalysis with hemianesthesia and hemianopsia, and also sometimes with hemiataxia. Such combinations are sometimes spoken of as the *leg-sensory* type, the *hemiplegic-aphasic* type, the *sensory-hemianopsic* type, the *hemianesthetic-hemianopsic* type, etc. The most common types of hemiplegia are due to capsuloganglionic lesions. Cortical lesions most frequently give rise to monoplegias, and to abortive, imperfect, and dissociated types of hemiplegia. After an attack of cortical hemorrhage or embolism, the patient, for instance, at first may show some involvement of face, arm, or leg, but most of the paralysis speedily passes off, leaving only a facial, an orolingual, a brachial, or a crural monoplegia, or one of the combined varieties of monoplegia. Occasionally in cortical cases the paralysis preponderates in the proximal portions of the limbs, although even in such cases some loss is generally present in the distal portions.

Often the condition left is one of hemiparesis (Fig. 306) rather than hemiparalysis, but in some part of the partially paralyzed member or face the loss of power will be found to preponderate. Temporary and partial aphasias often accompany cortical monoplegias, particularly of the facial and faciobrachial types. Hemiplegia occurs somewhat more frequently on the right side than on the left. Sixty cases studied in the Philadelphia Hospital show thirty-three cases of right and twenty-seven of left hemiplegia; and I believe that the percentage of a larger number of cases would prove to be higher than this in favor of the right side.* It was held by Brown-Séquard, and the opinion has been accepted by Bastian and others, that lesions of the right hemicerebrum are more severe and fatal than those of the left; but my experience has not taught anything of positive value regarding this question. Hughlings Jackson has expressed the opinion that double papillitis is more often associated with disease of the right than with disease of the left hemicerebrum. The illustration Fig. 307 shows the mechanism of hemiplegia from lesions in different parts of the cerebrum.

Fig. 306.

Hemiparesis with slight contractures of the hands; marked atrophy in the upper extremity, none in the lower; unilateral sweating on paralyzed side; loss of thermic sensation and allochiria; affection of nine months' standing.

Facial and Lingual Paralysis in Hemiplegics.—In the most common form of hemiplegia paralysis of the arm and leg is more complete than in the face, and when the latter is affected the lower portion is usually more decidedly paralyzed than the upper, all facial

* In 1895 Dr. Helen Baldwin, one of the internes of the Philadelphia Hospital, under my supervision made an examination of sixty hemiplegics, obtaining many interesting facts as to the side of the body affected by the paralysis; its completeness; its preponderance in the lower or in the upper extremity, or in the proximal or the distal portions of the limbs, and the association with the paralysis of partial or complete aphasia. Other observations were made on rigidity, contractures, athetosis or athetoid movements, and other spastic phenomena; on associated and substitutional movements; on tendon and muscle phenomena; on vasomotor conditions; on atrophy and other trophic changes; and on pain, hyperesthesia, and anesthesia. A study was also made of the non-paralyzed side. Reference will be made to this investigation in different portions of this section.

movements having a larger bilateral representation in each cerebral hemisphere than do those of the limbs. In rare cases of hemiplegia due

FIG. 307.

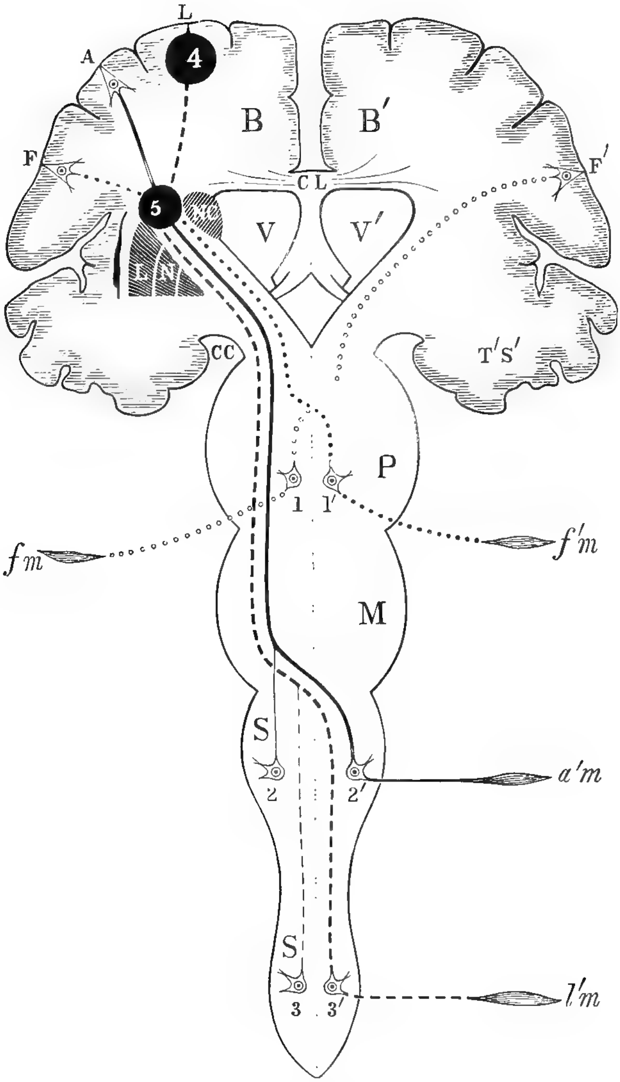


Diagram showing the arrangement of the motor tracts for both sides of the face, the left arm, and the left leg ; also the mechanism of hemiplegia from lesions in different parts of the cerebrum : B and B′, right and left hemicerebrums ; P, pons ; M, oblongata ; S, S, spinal cord ; V, V′, lateral ventricles ; NC, caudatum ; LN, lenticula ; CC, crus ; T′S′, left temporal lobe ; F, A, L, cortical centres for the face, arm, and leg in right hemisphere ; F′, cortical centre for the face in left hemisphere ; 1, 1′, right and left facial nerve nuclei ; 2, 2′, right and left nerve nuclei for upper extremity ; 3, 3′, right and left nerve nuclei for lower extremity ; *fm* and *f′m*, right and left facial muscles ; *a′m*, muscles of the left upper limb ; *l′m*, muscles of the left lower limb ; 4, localized lesion in the cortex, producing paralysis of the opposite leg ; 5, lesion of the pyramidal tract as it enters the internal capsule, producing hemiplegia on the opposite side of the body (paralysis of the face, arm, and leg). (Bramwell.)

to unilateral cerebral lesion the face is relatively as much paralyzed as the limbs. These cases can perhaps be accounted for in several ways. The control over the paralyzed side of the face may have been less normally than that over the other. When lesions destroy the region of the knee and the more anterior portion of the posterior limb of the capsule, the paralysis of the face and tongue will be more marked. Probably also lesions in the cephalic portion of the pons which destroy the tracts for the limbs, and the facial tracts just before their decussation, leave more decided loss of power in the face. On careless inspection a hemiplegic may apparently be able to use the paralyzed as well as the unparalyzed side of the face, when closer scrutiny will show some diminution of power. He may perhaps be able to close both eyes, but the eye of the paralyzed side will not be so tightly shut as the other, as shown by the lesser depth of the radiating furrows which indicate the contraction of the orbiculopalpebral muscle. After the zygomatic movements have largely recovered, the movements of the depressors and levators and of the orbicular muscle of the mouth and lips remain decidedly affected. These may be regarded as the most distal movements of the face. The tongue in hemiplegics shows many variations in the degree of paralysis. Sometimes it cannot be thrust out of the mouth at all, or it may at first be caught by the lips and teeth, the patient by a renewed effort getting it outside of the mouth. It may be deflected to one side or the other, and occasionally it tends to be retracted or to fall back into the pharynx.

Pathological Significance of Different Hemiplegic Types.—The type or subtype of paralysis presented in a case of chronic hemiplegia is largely dependent upon the position and extent of the original lesion, or at least of so much of it as persists. Early in the history of such cases the effects of pressure, of inhibition, and even of adjoining inflammation must be taken into account, but after the acute symptoms have subsided, and partial absorption has taken place, the lesion in a varying time acquires the dimensions which it will retain permanently unless another apoplexy occurs at the same site. A study of the diagrams Figs. 235 and 236, on pages 359 and 360, will serve to make clear some of the reasons for the variations in the distribution of the paralysis. In the majority of cases, as stated, the loss of power in the upper extremities preponderates over that in the lower, which is in accordance with the fact that the destructive effects of lesions are most frequently visited upon the middle portions of the posterior limb of the internal capsule, a region supplied largely by the lenticulostriate artery. A lesion situated more caudad may give preponderating leg paralysis, and this may be associated with anesthesia, owing to the involvement of the neighboring sensory tract. In the anesthetic-ataxic-hemianopsic type the lesion certainly involves the most posterior portion of the posterior limb of the internal capsule and the optic radiations. In some cases of this kind, as in

those recorded by Seguin, de Schweinitz, and the writer, the lesion is probably located at a position where the optic radiations and the internal capsule are in juxtaposition, and the thalamus may be involved in some instances. Differences in the site and extent of the lesion must not be regarded as the only or even the chief explanation of the differences in some of the clinical features of hemiplegia, as has already been sufficiently shown when discussing the reasons for the frequent preponderance of distal paralysis. Now and then a case of isolated capsular lesion has been put on record, as one by Parisot,—a case of facial monoplegia, the only muscle involved being the depressor of the left angle of the mouth. An old hemorrhagic cicatrix was found in the anterior part of the internal capsule, extending transversely across the lenticula just posterior to the geniculate fasciculus. Most frequently, so far as recorded cases show, limited palsies are of cortical origin, or at the most involve the cortex and the immediate subcortex.

Alternate Hemiplegias.—The *alternate hemiplegias* are types which will be more fully considered under diseases of the crus, pons, and oblongata. Often in lesions of the ventral portions of the pons the cranial nerves at or near their superficial origins are involved. In lesion of the lower ventral third, or conjointly of the ventral and lateral thirds, the alternate hemiplegia is of the arm and leg of the opposite side, and of the face on the same side; or facial, abducens, and auditory nerve paralysis may be present with contralateral paralysis of the extremities. In the middle of the ventral thirds, especially if the lesion extends laterally, paralysis of the leg and face of the opposite side may be associated with both motor and sensory paralysis in the distribution of the fifth nerve. In the cephaloventral segment the lesions may extend so as to implicate the crus and the third nerve, giving the oculomotor type of alternate hemiplegia; or some paralysis of both the third and fourth nerves may show on the same side.

Hemiplegia on the same Side as the Lesion in the Brain.—In the vast majority of cases the hemiplegia is contralateral to the brain lesion,—on the side opposite to the half of the brain which is diseased,—in accordance with well known anatomical and physiological facts. In rare instances, however, the paralysis is on the side of the lesion. Many years ago Brown-Séquard made a considerable collection of such cases, using them as arguments against the doctrine of cerebral localization, which was just beginning to find a firm footing. In my own experience I have known of only two cases confirmed by autopsy in which decided hemiplegia was present on the side of the lesion of the brain. One of these was an old case of capsuloganglionic softening, and the other was a case of recent hematoma of the dura. The usual explanation offered for these cases is the absence of the ordinary decussation of the pyramidal tract. Flechsig first and

a number of others more recently have shown that this decussation is liable to very considerable variations; but, as Gowers suggests, this explanation will not answer in all cases, as the decussation may take place below its usual position. Although such cases are rare, they may occasionally be of great practical importance; if, for instance, in the case of hematoma mentioned above, trephining had been decided upon, the operation would naturally have been performed on the side opposite to the paralysis, which would not have been that of the lesion.

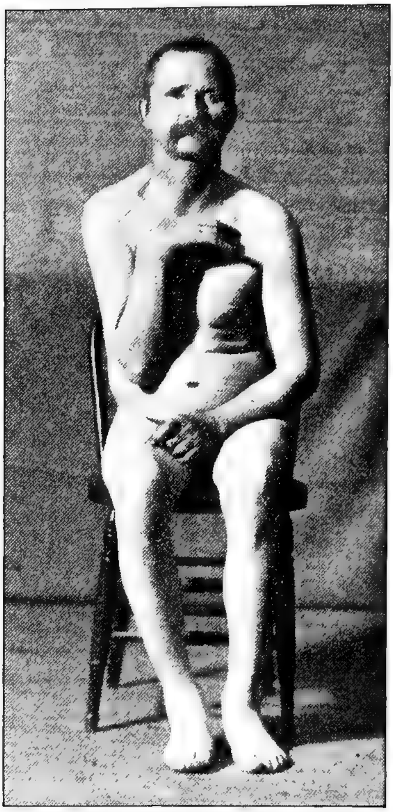
Disorders of Position and Movement in Hemiplegics.—The most important disorders of position and movement on the paralyzed side are contractures and rigidities; tremors, twitchings, and clonic spasms; athetosis and athetoid, choreic, and ataxic phenomena; and associated, substituted, and forced movements. While these need to be considered separately, it must not be forgotten that several of them may constitute parts of a condition which cannot be pathologically or clinically separated. Rigidity, contractures, coarse tremors, athetoid, choreoid, and ataxic phenomena, may all be closely amalgamated manifestations having a common origin.

Contractures and Rigidities.—*Flaccid and Spastic Paralysis.* The paralysis in hemiplegia may be of a flaccid or relaxed character, or it may present spastic or cramp-like features. The limbs may be from the first relaxed and totally helpless, a not unusual condition during the apoplectic attack. Instead of this flabby or flail-like paralysis, however, certain phenomena of spasm, contracture, and rigidity may be present in the limbs. Contractures and rigidities must be considered together, as they go hand in hand, and one may be and usually is dependent upon the other. The term contracture is applied to spastic muscular shortening; rigidity, to the more or less rigid and inflexible position in which the paralyzed extremity or any portion of it is held.

Characteristics of Hemiplegic Contracture. In contractured limbs flexions usually predominate over extensions, the result being certain peculiarities of attitude and gait. In some hemiplegics with decided paralysis in both the upper and lower extremities the contractures are pronounced and affect all parts of the limbs. The arm in some of these cases is drawn forcibly to the side, and the forearm, bent nearly at right angles to the arm, is carried over the chest and abdomen. Marked flexures are also commonly present in the wrist and fingers. The thigh is strongly adducted and sometimes slightly flexed; the leg is flexed on the thigh at a right angle or less. At the ankle and toes the contractures are also usually of a marked character. Irregularities in the contractures often cause grotesque positions to be assumed by the hand and fingers. In the illustrations Figs. 308, 309, and 310 various forms of contracture with rigidity are shown. Except when local structural changes are great, the contrac-

tures can be overcome by careful traction, the parts resuming their abnormal positions again when the force is removed. It is surprising how frequently the joints remain supple, allowing these contractures to be overcome by such persistent traction, even when strong contractures have persisted for months and years. The patients sometimes complain of a sensation of tension in the parts, and they can often be seen rubbing or working with the limbs to relieve this feeling. A contracture be-

Fig. 308.



Hemiplegia with strong contractures involving all parts of the affected limbs; marked atrophy in the upper extremity; right oculomotor paralysis, with left divergent squint and inward limitations; paralysis of nine years' duration.

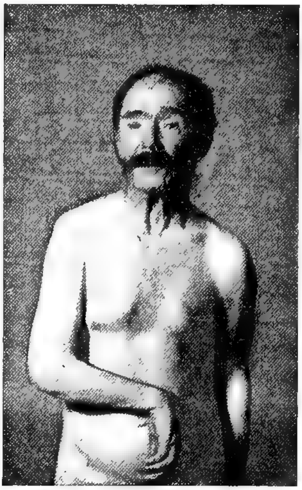
Fig. 309.

Hemiplegia with partial aphasia; marked contractures of the shoulder, wrist, and hand; marked atrophy on paralyzed side; paralysis of thirty-five years' duration.

ginning in one set of muscles tends to be diffused to associated muscles, or to become general through the limb. The contractures disappear under ether or chloroform narcosis. Usually in all cases in which marked contractures are present the tendon and muscle phenomena are exaggerated. Besides true rigidity and contracture, the neuromuscular apparatus is often in a state which Brissaud has described as one of "imminence of contracture,"—ready to contract or to exhibit other spastic phenomena on the slightest provocation.

Contractures were present in forty-three out of the sixty cases at the Philadelphia Hospital, and these were in the upper extremity alone in about two thirds of the cases. In rare instances contractures of the face are present in old hemiplegics; but both contractures and twitchings are more frequent and more diagnostic in peripheral facial paralysis than when the affection is part of a hemiplegia and due to a central lesion.

Fig. 310.



Hemiplegia with marked contracture at elbow and wrist; paralysis of several years' standing.

Mechanism of Secondary Contractures. The occurrence of secondary contractures has been variously explained. They have been attributed simply to retraction of the muscles and of the soft parts, a manifestly insufficient explanation. The spreading of motor impulses from the healthy to the paralyzed side, exaggeration of muscle tonus, and permanent muscular activity due to the irritable condition of the spinal cord are among other insufficient hypotheses which have been advanced. According to Marie, the pyramidal bundle plays a rôle of arrest, analogous to that exerted by the pneumogastric on the heart; and, as one mission of the pyramidal bundle is to restrain and regulate the gray matter of the bulb and cord, contracture takes place and persists because the inhibitory action of the higher cerebral centres is impaired by the destruction of the fibres of this bundle.

Rigidities and Pseudocontractures. The rigidity of the paralyzed limbs may be either *early* or *late.* One form of early rigidity is sometimes designated *initial* rigidity, which comes on at the time of the apoplexy and may pass away in a few hours. A more persistent early rigidity usually develops a few hours after the beginning of the apoplectic attack, and lasts for several days or weeks. The initial rigidity, which is probably dependent upon the immediate irritating effects of the apoplectic seizure, is frequently absent. Initial rigidity may pass into early rigidity, and the latter may merge into late rigidity. Late rigidity, however, commonly shows itself after several weeks, the paralysis having been up to the time of its appearance of a flaccid character, or having shown only a slight tendency to contracture. When contracture and late rigidity have persisted for a long time, certain changes take place in the parts affected, these being in part due to fixity of position and disuse, and in part to central degenerative changes; but in my own experience this *structural* rigidity is rare even in cases of long duration. All forms of rigidity are, as a rule, more marked in the arm than in the leg, and in the distal rather than

in the proximal portions of the limbs. In the development of spasmodic contracture, in certain cases retraction of the tendons, probably due to rupture and cicatrization of tendinous fibres, results in malformations. Pseudocontractures are observed as the results of traumatisms and inflammations of the muscles. In Parkinson's disease, and in primary amyotrophies, contractures are characterized clinically by special rigidity to touch, by their irregular distribution, by the absence of any tendency to become general, by the nonexaggeration of the reflexes, and by their nondisappearance during chloroform narcosis. Ischemic pseudocontracture follows prolonged deprivation of arterial blood, is characterized by coldness and rigidity of the limb, and is due to a morbid change in the muscular fibres. (Blocq.)

Associated and Substituted Movements.—The term associated movement, as used in describing hemiplegic phenomena, is commonly applied to an involuntary movement in one part as a result of a voluntary movement in another. Substituted movements are those which take the place of movements that the patient intends or tries to perform. Associated and substituted movements can be conveniently subdivided into (1) movements in the muscles of the nonparalyzed side, with or instead of an intended movement; (2) similar movements in paralyzed muscles; and (3) involuntary original movements with involuntary associated and substituted movements. (Senator.) These associated movements are most frequently observed in the upper extremity. Twenty-five of the sixty hemiplegics studied in the Philadelphia Hospital showed associated or substituted movements. In ten of these cases volitional movements on the unaffected side caused involuntary movements of the paralyzed side, and in eleven cases movements or attempted movements of the paralyzed limb caused involuntary movements on the other side; while in four cases the movements were of special character. Nearly all the results were obtained from the upper extremities. In one case, however, attempts to use the paralyzed arm caused movements in the paralyzed leg. In another case coughing caused the paralyzed hand to be moved to the face. In a third yawning was followed by an extension of the paralyzed fingers and raising the hand towards the head; and in a fourth yawning or taking deep breaths caused the forearm of the paralyzed side to be flexed, the index finger to be extended, and the lower extremity to be extended and to become rigid. Beer has recorded a case of hemiplegia with contractures of the arm in which passive movement was almost impossible. When the patient extended his right (sound) arm, the movement was slight; but the result of a yawn was surprising. The contracted muscles relaxed so completely that extreme dorsal flexion of the wrist and fingers occurred, while the arm was lifted and abducted, the shoulder being almost rigid before. Similar results were obtained in a second

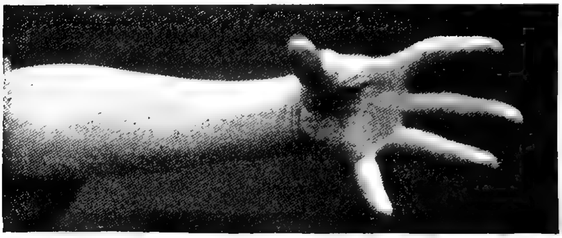
case. Various explanations of the origin of associated movements have been suggested. Increased excitability of the centres and diminished resistance of the tracts connecting the centres through which these movements are produced, transference of reflexes, the overflow of normal impulses, and the loss of inhibitory power, have been evoked in their explanation. The structural connections through which these movements are effected can be understood by what has been already stated as to the splitting of the fibres which go to the lateral pyramidal tracts, and the probable commissural connections between the centres of both sides of the spinal cord. As Senator has shown, the cause of associated movements may be situated altogether outside of the central nervous system. One of his patients had received a compound fracture of the skull twelve years before coming under observation, and one year after had become hemiplegic and aphasic. After the fracture, on swallowing he experienced pain in the angle of the jaw upon the right side. Excitement and irritation brought on choreic movements. On protruding the tongue, the paralyzed arm became flexed, the index finger extended, the others clenched, and the hand was raised to a level with the ear as in a military salute. The same movements were produced by pressure along the painful area at the angle of the jaw, and along the anterior edge of the upper third of the sternomastoid muscle. Senator believed this to be due to an inflammatory infiltration around the nerves of the cervical plexus, extending up to the root of the tongue, regarding the movement as reflex and not due to direct irritation. Associated movements may result from cutaneous stimulation, as in a case recorded by Nothnagel, in which moderate pinching of the paralyzed arm caused muscular contraction in the sound arm ; when the pinching was a little stronger the contractions extended to the sound leg ; and when the irritation was still stronger, to the leg on the hemiplegic side. The limb pinched always remained quiet.

Forced Movements.—Forced movements, or movements which the patient tends to make in spite of his will, are not often seen among chronic hemiplegics, and when present are commonly indicative of unusual irritation at the site of the lesion. They may be of peculiar character according to the location of the lesion, as will be understood by reference to what has already been said about the effects of destruction or irritation of the cerebellum or cerebellar peduncles. They are occasionally seen during the early stage of hemorrhagic apoplexy, but are more common in tumors. They may be of a violent and almost uncontrollable character. Ewald reports a case of disseminated miliary tuberculosis in a boy, in which the patient showed decided jerking of the left arm and shoulder, so that he was compelled to hold the left arm forcibly against his side with the right to keep it still. When the left arm was released, it was immediately extended backward from the shoulder at an angle of

forty-five degrees, and was affected with jerkings in all its joints. A solitary tubercular mass, about an inch in diameter, was found in the right thalamus, surrounded by a red ring of inflammation or degeneration, involving a part of the posterior limb of the internal capsule. Forced movements have been attributed to unilateral incomplete paralysis; to excessive activity of one side of the body the result of unusual central stimulation; and to vertigo and giddiness. Conjugate deviation of the eyes and rotation of the head constitute one form of forced positions and movements.

Athetosis and Athetoid Phenomena.—The word *athetosis,* which is derived from a Greek word meaning "without fixed position," is applied to a peculiar form of grotesque recurring mobile spasm sometimes observed in adult hemiplegics, although when organic and of cerebral origin it is usually congenital or the result of an affection occurring in early childhood. It is a somewhat frequent phenomenon in the cerebral palsies of children, in connection with which it will be again considered. Strictly speaking, it is not a disease, but a part of a symptom group. In hemiplegics it may be dependent upon lesions of the motor tract. In Fig. 311 are shown the arm and hand of a patient who suffered from a marked form of athetosis on the paralyzed side. In the section on the cerebral palsies of children other illustrations of athetosis are shown in several figures. In adult hemiplegics athetosis or athetoid mobile spasm is nearly always associated with a greater or less degree of permanent rigidity with contracture.

Fig. 311.



Position of the hand and fingers in one of the forms of athetosis.

Tremors, Twitchings, and other Abnormal Movements.—Tremor in the paralyzed limbs is not common, but in rare instances a tremor somewhat similar to that of paralysis agitans has been observed, as in a case reported by Bastian in which the patient was affected with tremors in the right hand and wrist after she had regained some motor power. The lesion was probably situated in the crus. Twitchings of various kinds are frequently observed, and are sometimes readily induced by handling the limb or by the unavailing efforts of the patient to use it. Rhythmical movements, such as alternate flexions and extensions, are occasionally noted, and are almost exclusively confined to the upper extremity. Choreoid and ataxic movements are also occasional phenomena; and a posthemiplegic chorea has been described, but the movements in these cases

are not strictly of a choreic character. They are usually a commingling of a clonic spasm and of tremulous, choreoid, athetoid, and forced movements. Movements of this kind were present in eleven out of sixty cases. It is interesting in this connection to note the curious effect that a hemiplegia may have on the tremor of insular sclerosis. Sinkler has recorded a case of insular sclerosis in which, as the result of an apoplexy, the intention tremor disappeared on the paralyzed side.

Cutaneous Reflexes.—During the apoplectic period the cutaneous reflexes, as a rule, are abolished, and if the patient recovers to become a chronic hemiplegic they may be absent or much diminished for a long time or altogether. The cremasteric and abdominal reflexes in particular may be absent, even if the plantar reflex is present or has returned after it has been lost for a time. The early loss of cutaneous reflex action is occasionally important from a diagnostic point of view. The explanation of the diminished or abolished skin reflexes is somewhat difficult, particularly in the light of the fact that muscle and tendon phenomena are usually exaggerated, suggesting the idea that the mechanisms which control the two sets of phenomena are different. It has been held by some that the spinal centres are controlled by lower encephalic centres, and the latter by centres still higher, and that the abolition of the cutaneous reflexes is due to the effect of lesions which prevent the inhibitory influence exerted by the higher upon the lower encephalic centres. In rare instances the cutaneous reflexes are increased.

Tendon and Muscle Phenomena.—As a rule, knee jerk, muscle jerk, and tendon and muscle phenomena in the upper extremity are exaggerated on the paralyzed side; the front tap phenomenon and ankle clonus can often be readily elicited, and the toe jerk also in rare instances. In some cases the entire paralyzed limb can be thrown into grotesque twitchings, jerkings, or oscillations by slight percussion over the muscles, tendons, or bones, or such local spasmodic phenomena may occur spontaneously, or may seem to be of spontaneous origin, being in reality due to accidental irritation of the parts. In rare cases the muscle and tendon phenomena may be below the normal or even absent, and in these the question of cerebellar disease or of lesions somewhere outside of the cerebrum needs to be taken into consideration. The following results were obtained for the paralyzed side in the sixty cases examined by Dr. Baldwin:

Knee jerk exaggerated	45	cases.
Knee jerk absent	4	"
Muscle jerk exaggerated	43	"
Muscle jerk absent	5	"
Ankle clonus present	20	"
Front tap present	35	"

Sensory Disorders.—The sensory disorders of hemiplegics may be (1) pain and hyperesthesia; (2) paresthesia; and (3) anesthesia, local or of one half of the body. The perversions may be of one or of several of the different varieties of sensation.

Pain and Hyperesthesia. Pain of a severe character may precede an apoplectic attack which results in hemiplegia, may occur during such an attack, or may follow the attack, constituting part of the hemiplegic state. Weir Mitchell has described what he calls prehemiplegic and posthemiplegic pains. In rare cases acute pain in the musculature of the affected side is an immediate prodrome of an attack of hemiplegia, being present from twenty-four to forty-eight hours before the occurrence of the attack. Mitchell records the case of a woman who was seized with violent pain in the leg and arm without joint lesions, and who within thirty-six hours had a quite complete attack of hemiplegia on the same side, after which the pain soon faded away. In some cases unilateral fibroid pain and soreness may be persistently present, or may recur at short intervals for a long time, before the hemiplegic state, and similar pains may be present after the patient has become hemiplegic. These pains are often associated with the joint disorders to which attention is called in another paragraph. A true neuritis, either of accidental origin or a part of the organic affection, may be present in hemiplegics. In some of the instances in which pain has been present it was caused by a subinflammatory hypertrophy of the nerves and their sheaths. Hyperesthesia and tenderness are sometimes to be explained in this way; in other cases the pain and paresthesia are probably of cortical or cerebral origin. In sixty cases pain was complained of in thirty-one, and in eighteen of these it was in both upper and lower extremities. Hyperesthesia was present in ten cases.

Paresthesias. Paresthesias or perverted sensations of various kinds are somewhat common in hemiplegics, who frequently complain of feelings of numbness, burning, itching, heaviness, deadness, etc., in the paralyzed half of the body. These symptoms are sometimes local in origin, due to nutritive or inflammatory changes in the limbs, as the result of disease or of accident. Doubtless also they are sometimes due to the cortical or subcortical disease. As Bremer has shown, pain, itching, and other sensations may be of cortical origin.

Painful Nodes. Painful periosteal nodes are now and then observed in hemiplegic limbs, and attention has been called to them by Weir Mitchell. He records the case of a man, forty-five years old, who two or three months after an apoplectic attack developed joint lesions with rigidity and contracture. An elongated tender node about an inch wide was found three inches above the ankle, and at the insertion of the deltoid another one still more prominent. No history nor evidences of syphilis could be discovered, and the patient, who was a plumber, showed no signs of lead poisoning.

Anesthesias. Hemianesthesia of organic origin without loss of power on the affected side is of very rare occurrence, but is occasionally seen, and is commonly due to a limited lesion of the most posterior portion of the posterior limb of the internal capsule, or to a lesion of the thalamus. Recent investigations have brought neurologists back to the view largely held in former years, and especially by the British school, that the thalamus is a sensory organ; nevertheless, the theory that a portion of the sensory tract is contained in the hinder limb of the internal capsule cannot be regarded as overthrown, or as necessarily inconsistent with the view that the thalamus is the basal terminus of many of the incoming sensory tracts. A majority of cases of chronic hemiplegia do not show any impairment or

FIG. 312.

Diagram showing the area of retained sensation on the face in a patient otherwise totally hemianesthetic; anesthesia shown by the shading; hemiplegia and hemianesthesia due to a destructive lesion of the thalamus and posterior extremity of the internal capsule.

disorder of sensation even on close investigation, although such sensory disturbances are more frequent than is supposed, and are sometimes overlooked. In all cases an examination should be made for perversions or losses of the different varieties of cutaneous and muscular sensation—for changes in tactile, pathic, and thermic sensibility, and in the senses of pressure, locality, and all the varieties of sensation which enter into the so-called muscular sense. Hemianesthesia may be present with extensive paralysis of the leg, arm, and face, or the paralysis may preponderate in the leg, giving the leg-sensory type. An anesthetic area confined to one limb or to one part of it is occasionally discovered, but such cases are rare. It should be understood, however, that all such cases are not of hys-

terical origin, even when they are not confined to particular nerve distributions. Savill has reported several cases which apparently demonstrate that lesions of the gyrus fornicatus and its immediate subcortex will cause more or less limited anesthesia of the opposite extremities according to the exact site and dimensions of the lesion (see page 341). What is supposed to be a complete hemianesthesia sometimes on examination proves to be incomplete. In one of my cases of lesion of the thalamus and internal capsule the patient was totally anesthetic over one half of the body, except in one limited region of the face. This area of retained sensation included the orbital region, the side of the nose, and the left cheek within a line drawn from the external angular process of the frontal bone to the ala of the nose. The mucous membrane of the mouth was sensitive in an area which corresponded, so far as could be determined, to the cutaneous region of retained sensation. (Fig. 312.) In organic cerebral hemiplegias some forms of sensation may be lost and others retained, although such cases are rare. Some form of anesthesia was present in five out of sixty cases. Occasionally hemiplegics show a loss of the sense of position of the limbs. This symptom is not common, and is usually associated with hemianesthesia.

Disorders of the Special Senses.—Disorders of hearing, smell, and taste are not common in hemiplegia, but are observed in special cases which are to be explained by unusual extensions of the lesions causing the paralysis to the tracts and centres for these special senses, or by the presence of two or more lesions. A hemiparesis with cerebral deafness and paraphasia may be caused by a large lesion of the temporal lobe, the loss of power being due to pressure on the motor region or tracts, and the auditory disturbances to the destructive effects of the lesion. Affections of sight are of more frequent occurrence, and especially hemianopsia, which is present in one of the types to which attention has already been directed, in which the symptom complex is hemiplegia or hemiparesis with hemianesthesia, hemiataxia, and hemianopsia. Hemianopsia will be further considered when affections of the optic nerve and its correlated encephalic structures are discussed. Besides hemianopsia, other disturbances of vision are occasionally present, such as central amblyopia, phosphenes, and visual hallucinations.

Vasomotor, Secretory, and other Allied Phenomena.—The vasomotor changes in the paralyzed limbs may be of varying, and even of opposite, character. In the early period of hemiplegia the limbs may be warmer, and may present a somewhat hyperemic appearance. Later the parts usually become colder and are often purplish or livid. Edema is occasionally present in the paralyzed extremities, and is usually confined to their proximal portions. It may be an early phenomenon, coming on during the apoplectic attack, and disappearing largely or altogether as the attack passes off. As

chronic nephritis is a comparatively common accompaniment of apoplexy and hemiplegia, the edema or anasarca of renal disease may also be present. When such renal disease is present, the edema may be more marked in the paralyzed extremities. Edema with glossy skin is sometimes observed over a large portion of the limbs; and in these cases pain and hyperesthesia, with other evidences of neuritis, are usually also present. Edema was present in seven out of our sixty cases, and local or unilateral sweating also in seven cases.

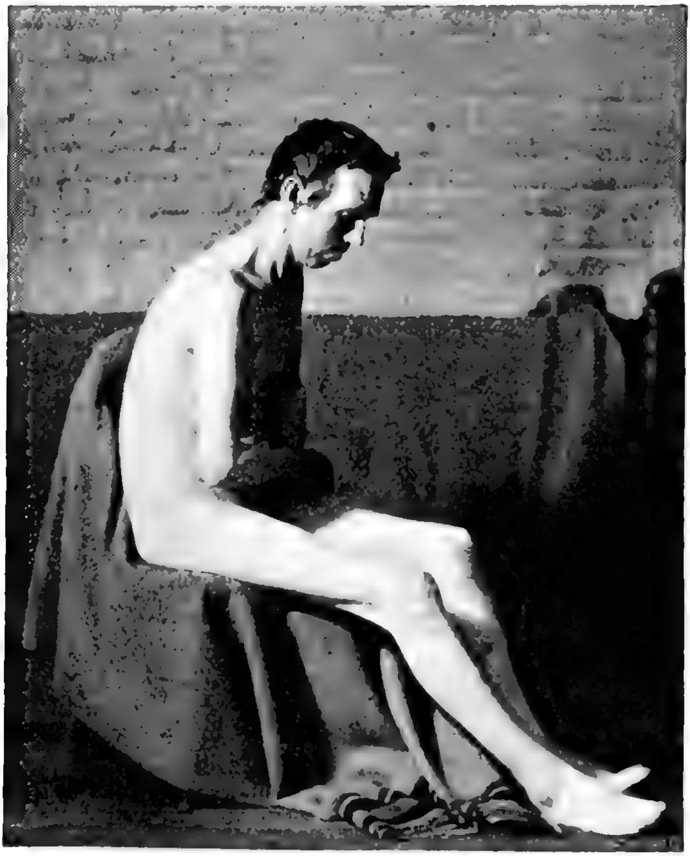
Surface Temperature.—In the apoplectic period both axillary and surface temperatures are usually somewhat increased on the paralyzed side, but after a lapse of two or three weeks the temperature will be found to be decreased on this side, and in chronic hemiplegics the surface temperatures are almost invariably found to be less on the paralyzed than on the nonparalyzed side. A careful examination of the surface temperature in sixty cases was made by Dr. Baldwin. The average temperature on the sound side was 95.4° F., and on the paralyzed side it was 93.5° F. The actual temperatures taken in thirty of these cases are given in the following table:*

Case.	Nonparalyzed Side.	Paralyzed Side.	Difference.	Duration of Disease.
1.	95.4	91.8	3.6	3 years.
2.	93.8	93.4	.4	3 "
3.	96.5	95.8	.7	4 "
4.	95.2	91.0	4.2	4 "
5.	93.5	92.3	1.2	4 "
6.	96.2	91.5	4.7	5 "
7.	93.8	93.8	. .	5 "
8.	96.3	96.1	.2	6 "
9.	95.7	95.2	.5	6 "
10.	96.9	96.0	.9	6 "
11.	94.8	93.6	1.2	6 "
12.	95.0	94.1	.9	6 "
13.	95.6	86.8	8.8	7 "
14.	95.2	93.4	1.8	7 "
15.	95.0	92.8	2.2	7 "
16.	94.0	92.4	1.6	7 "
17.	94.5	93.8	.7	8 "
18.	96.5	94.9	1.6	9 "
19.	96.0	96.0	. .	10 "
20.	95.6	92.0	3.6	10 "
21.	93.2	91.2	2.0	11 "
22.	93.6	93.6	. .	13 "
23.	94.9	93.5	1.4	15 "
24.	93.2	87.5	5.7	15 "
25.	94.7	92.5	2.2	18 "
26.	96.0	96.0	. .	18 "
27.	94.5	93.0	1.5	20 "
28.	93.7	91.7	2.0	23 "
29.	93.0	92.5	.5	25 "
30.	95.8	94.0	1.8	35 "

* The thermometers used were carefully tested. They were of the flat spiral bulb variety and most approved pattern. A number of preliminary trials showed that they never continued to register after the lapse of five minutes. The thermometer was in all cases placed over the supinator longus muscle.

Hemiplegic Atrophy.—In hemiplegias and monoplegias which are the result of acute lesions occurring after adult life has been reached, wasting of the affected limbs and face is not so marked as when the lesion is congenital or originates in infancy and early childhood. At one time it was thought that atrophy was very seldom present in adult cerebral palsies; but a careful study of a large number of hemiplegics shows that some atrophy is not infrequent, and usually it is more marked in the distal portions of the limbs.

Fig. 313.



Hemiplegia with aphasia, contractures, and marked atrophy on the paralyzed side. (Dercum.)

The little muscles of the hands, and the muscles of the forearm particularly, often suffer. Occasionally atrophy is quite noticeable in the muscles of the foot and face. It is more than the diminution in bulk due to disuse. These atrophies are to be attributed to degenerations of the pyramidal tracts and involution of the anterior horns, and to degenerations subsequent to peripheral neuritis occurring in the paralyzed limbs. The atrophy may in some instances be due

to the destruction of trophic centres in the cerebrum. Savill, for example, has suggested on the basis of a clinicopathological observation that such centres may be situated in the limbic lobe. An illustration of a case of hemiplegia with well marked atrophy is given in Fig. 313. Moderate atrophy of the arm is also exhibited in Fig. 314; and in other illustrations already given more or less decided atrophy appears (Figs. 306, 308, 309). In all cases in which hemiplegia occurs at a very early age, after a few years the entire paralyzed half of the body presents an arrested or atrophied appearance. These cases will be more fully considered under infantile cerebral palsies.

Fig. 314.

Hemiplegia and aphasia, with some atrophy of the right arm; no contractures.

Atrophy was present on the paralyzed side in thirty-five of sixty cases, being marked in the seventeen cases shown in the following table.

Side Paralyzed.	Forearm just below Elbow.			Middle of Leg below Knee.		
	Sound.	Paralyzed.	Difference.	Sound.	Paralyzed.	Difference.
Right.	11.00	9.50	1.50	14.50	14.25	.25
Right.	10.00	8.00	2.00	No difference.		
Left.	10.50	9.50	1.00	"	"	
Right.	10.50	9.50	1.00	"	"	
Left.	10.00	9.25	.75	12.25	11.50	.75
Left.	9.50	9.00	.50	No difference.		
Left.	8.25	6.75	1.50	12.25	11.00	1.25
Left.	10.50	9.25	1.25	No difference.		
Left.	9.25	8.25	1.00	13.50	12.50	1.00
Left.	10.00	9.25	.75	No difference.		
Right.	8.50	7.50	1.00	"	"	
Right.	13.00	10.00	3.00	"	"	
Left.	9.00	8.00	1.00	12.00	10.00	2.00
Right.	10.50	9.50	1.00	14.00	12.00	2.00
Left.	10.00	9.50	.50	No difference.		
Right.	10.00	9.00	1.00	13.00	12.50	.50
Right.	10.50	9.00	1.50	No difference.		

Measurements in inches.

Miscellaneous Trophic Phenomena.—During grave apoplectic attacks gluteal eschars or bedsores sometimes form on the paralyzed side. They were first particularly described by Charcot, who regarded them as most inauspicious signs, the cases in which they were present often terminating fatally. When the patient recovers, such eschars are troublesome for a long time. During the apoplectic attack, and sometimes later, the skin in exposed positions may become sore, and it sloughs more readily than on the nonparalyzed side. Sometimes the trophic and other eschars form late in the history of the hemiplegia, when the patient is stricken in his last illness by a recurrence of the cerebral trouble or by some intercurrent or concomitant affection, as pneumonia, nephritis, or endocarditis. Chronic hemiplegics may also show a tendency to ulcerations and other trophic disturbances in the paralyzed limbs. The skin may be thickened or scaly, or in rare instances it may be smoother and finer than on the paralyzed side. The occurrence of a furunculous eruption limited to the paralyzed side of the face has been noted in hemiplegics. Changes in the nails, such as groovings and other markings, brittleness, roughness, increased thickness, peculiar incurvations and twistings, and alterations in color, often to a yellowish hue, are somewhat common. The nails may show a tendency to different rates of growth on the paralyzed side and on the nonparalyzed side. These changes can readily be observed on the finger nails, and are often especially marked on the nails of the great toes. Some abnormalities of the nails were observed in more than half the cases studied in the Philadelphia Hospital. The growth of the hair may be really or apparently increased, and it may be of a coarser character than on the healthy side. The alterations both in nails and hair may be due to the local neuritis which is so often present in the limbs, and to which attention has been called when speaking of pain and hyperesthesia, rather than to the disease of the brain. It is well known that in various forms of neuritis and in spinal disease unassociated with encephalic lesions such trophic changes are present. Due consideration, however, must be given to encephalic trophic centres both cortical and basal.

Joint Affections.—Joint affections or arthropathies, like painful disorders of the limbs, may precede an attack which causes hemiplegia, or may be manifested at various periods in the history of the hemiplegic. Weir Mitchell records cases in which repeated attacks of tenderness and swelling of the joints were present, which were strictly limited to one side, this side subsequently becoming paralyzed. In such cases the lesions in the joints sometimes increase after the palsy is established, and may persist as chronic affections. In other cases joint lesions follow closely on the apoplectic attack. Mitchell mentions four cases in which, with right sided cerebral lesion, such affections of the joints followed within four days. In other cases

arthropathies occur at irregular periods in the history of the hemiplegic. These joint affections are not to be dismissed simply as instances of unilateral rheumatism, for while it is of course possible for a true hemiplegic to be attacked with rheumatism, it is uncommon. More commonly joint inflammations appear in from three to six weeks after the apoplectic onset. They are referred by some to the period of inflammatory reaction. The arthropathies are in some cases to be referred to hypertrophic neuritis, and in others to the influence of central lesions; or both causes may sometimes be operative. When arthropathies occur during the acute stage of an apoplexy, while any joint may be selected, the articulations most frequently affected are the shoulder, wrist, elbow, those of the hand and fingers, the knee, and the foot. Severe arthropathies, like the gluteal eschars with which they may simultaneously appear, are often of evil omen. In chronic hemiplegia they may be evidences of local trouble, or they may develop with a fresh apoplectic attack. Autopsies have shown that the joints possess all the characteristics of a subacute synovitis. In some instances the articulatory affections are so slight as almost to escape notice.

Paralysis of the Sphincters.—During the apoplectic state involuntary evacuations of feces and urine are the rule. If, however, the patient recovers from the attack to become a chronic hemiplegic, control over the sphincters is usually regained, although it is often weakened. Occasionally the incontinence of urine continues for weeks or months, and it may be a persistent symptom. In large lesions of the cerebellum loss of control over the sphincters is somewhat common. Frequent seminal emissions with imperfect erections, and failure in coition, are sometimes present for many months after the onset of a hemiplegic attack. Sexual desire and power may be permanently impaired.

Disorders of Mentality.—While chronic hemiplegics often retain a large amount of intellectual energy and ability, some weakening or disturbance of mentality is comparatively common. This may be influenced by the age of the patient, by his previous intellectual vigor, or by the presence of chronic disease. Patients advanced in life and those who are prematurely senile usually show the most marked changes. After a second or a third attack the mental deficiency and disturbance become more decided; they are especially marked when the lesions are multiple, and when large areas of the cortex are implicated. The destruction of important subcortical associating tracts also gives rise to confusion and slowness in mental action. If the prefrontal lobe is involved, the psychical disorders which are present may have the special characteristic already detailed. Seguin believed that left hemiplegics showed a greater tendency to emotional disturbances than right hemiplegics. In almost all hemiplegics more or less emotionality on the one hand, or an

indifferent and apathetic state on the other, is after a time present. The emotional manifestations may be indirect in origin. The patient, finding himself helpless or handicapped, is often restive under his hard yoke. Disturbances of speech may mask or confuse his real mental state.

Phenomena on the Nonparalyzed Side.—Interesting clinical phenomena are to be noted on the unparalyzed side, these corresponding with what would be expected in the light of what has been said regarding bilateral secondary degenerations. As early as 1875 Westphal noted that in some hemiplegics foot clonus could be elicited on the healthy side. Dignat has shown that muscular force is often diminished fifty per cent. in the lower limbs, and also in the upper, but not to the same extent. The healthy limbs suffer partly because of the disruption of coordination between the two sides of the body. Friedländer observed that the diminution of strength in the left upper limb was greater in right hemiplegics than that of the right in left hemiplegics. In the late stages of a long continued hemiplegia, especially if the patient is advanced in years, the loss of power may be so great that the patient is almost paraplegic. More or less incoordination is commonly present. All the tendon and muscle phenomena may be exaggerated on the healthy side, although not to the same extent as on the affected side. Even a species of contracture or pseudocontracture may be present, and forced movements are sometimes observed. The following are a few of the points regarding the nonparalyzed side brought out by a study of the sixty cases in the Philadelphia Hospital:

Considerable loss of power present	31 cases.
Increased muscular irritability	7 "
Tremor, slight clonic spasm, or choreoid movements	7 "
Associated movements on nonparalyzed side from movements on paralyzed side	14 "
Associated movements on paralyzed side from movements on nonparalyzed side	8 "
Knee jerk increased	16 "
Knee jerk abolished	2 "
Muscle jerk increased	15 "
Muscle jerk abolished	2 "
Ankle clonus present	6 "
Front tap present	11 "

Bilateral or Double Hemiplegia.—A bilateral or double hemiplegia is occasionally observed in adults, and may be due to successive or simultaneous lesions of the same character as those which produce ordinary hemiplegia. The diplegias of children constitute a special class, to which no reference is made here. A bilateral case may simply give on both sides the clinical features which have been described for the ordinary forms of hemiplegia. The patient is likely to succumb to the attack which duplicates his paralysis. In a special

form of bilateral hemiplegia described by various writers, the glosso-labial syndrome is present and prominent, the affection constituting one of the forms of so-called pseudobulbar paralysis. The lesions may be cortical, chiefly occupying the lower extremities of the central convolutions, where are located the centres for the lips, tongue, larynx, masticatory muscles, and pharynx. They are, however, sometimes intrahemispheric, and in reported cases have been found chiefly in the inferior part of the putamen, or external segment of the lenticula; but in some cases also they have been present in the orolingual and speech tracts. The predominating symptoms in these cases are disturbances of the organs of articulation, associated with greater or less degree of paralysis in other parts. Some reference has already been made, on page 353, to pseudobulbar paralysis. In exceptional cases a unilateral lesion has been sufficient to cause the bilateral pseudobulbar syndrome. In these cases one hemisphere may have sufficed in the particular individual for the representation of the bilateral movements affected, the corresponding centres in the other hemisphere having had little or no functional importance.

Diagnosis.—*Etiological Diagnosis.* The etiological diagnosis of a hemiplegia or monoplegia may be of the highest importance. The etiological varieties of hemiplegia can be classified under those due to organic diseases of the nervous centres; those due to intoxications and infections; and those associated with the so-called neuroses. The organic nervous affections include traumatic and spontaneous cranioencephalic lesions, focal diseases of the brain, and some of the cerebrospinal degenerations. Traumatisms, meningeal hemorrhages, pachymeningitis, the different varieties of leptomeningitis, hemorrhage, softening, tumor, and abscess, have already been considered. Hemiplegia may also occur in the course of such cerebrospinal diseases as tabes, disseminated sclerosis, and paralytic dementia. Some of the intoxications of which hemiplegia may be the result are uremia, diabetes, alcoholism, lead poisoning, and other metallic or gaseous poisonings. It may accompany or immediately follow such affections as pneumonia or pleurisy, malarial, typhoid, and eruptive fevers, diphtheria, and the puerperal state; or it may be a concomitant of such chronic infections as syphilis and tuberculosis. Hemiplegia or hemiparesis may also be present in general affections of the nervous system, like paralysis agitans, hysteria, and chorea. It is clear that the diagnosis of hemiplegia in many of these affections will confront the physician at the time of its onset or during the acute stages of the disease of which it is a part. It is only necessary to mention here these numerous etiological forms of hemiplegia, in order that the physician may be on his guard in studying the more usual types which have just engaged our attention.

Points in the Diagnosis of Chronic Hemiplegia. As a hemiplegic often comes under the physician's notice after the case has become chronic,

the pathological cause of the paralysis may be overlooked or misunderstood. Both in hospital and in private practice old hemiplegics are often passed by as hopeless, it being taken for granted that the syndromes presented are due to old hemorrhagic or embolic cysts, when in fact a tumor, an abscess, or some other lesion more or less amenable to treatment may be present. The mistake is the more likely to be made because tumors, so far as paralysis is concerned, may remain latent for a long period, and because the paralysis may come on suddenly with an apoplectic seizure, the result of the intercurrent hemorrhage or of the rapid breakdown of the tissue near the growth. Abscesses may also remain for a long time latent. In every case, therefore, a careful study should be made of the type of the paralysis, and of the concomitant symptoms and conditions. The ophthalmoscope may be of great service by showing that papillitis is or is not present. Septic symptoms may point the way to the diagnosis of a chronic abscess. Diffuse syphilis of the brain or general paralysis of the insane may at times present features closely resembling those of some of the usual or unusual types of hemiplegia due to focal lesions. The attitude and gait of a hemiplegic may vary considerably, but in the usual types it is often characteristic. The patient carries himself somewhat towards the sound side, projecting the paralyzed limb forward, the paralyzed foot and leg being often made to describe the arc of a circle. This characteristic gait was first described by Todd. Charcot suggested calling it the *helicopod* walk, from words meaning to walk in a half circle; in contradistinction to the *helcopod* walk or dragging gait of hysterics. In some organic cases, however, the leg is also distinctly dragged.

Diagnostic Importance of Jacksonian Epilepsy and General Convulsions. In chronic as in acute hemiplegia, either Jacksonian epilepsy or general convulsions are occasional features. Local or Jacksonian epilepsy is characterized by an admixture of clonic and tonic spasm in groups of muscles related to definite centres and areas of the cortex, these being commonly characterized by signal symptoms and a serial order in the motor phenomena (see pages 351, 352). The spasm often shows a tendency to diffuse to many muscles of the same side, and less frequently to those of the other half of the body. The epilepsy may be either facial, brachial, or crural in type. The first gives a unilateral spasm which spreads from the face to the upper and then to the lower limb; in the brachial type the spasm diffuses from the upper limb to the face and next to the lower extremity; while in the crural type the most common order of diffusion is from the lower limb to the upper, and from the latter to the face. Not only the nature but the probable location of the lesion may be indicated by such spasm. The occurrence of Jacksonian epilepsy frequently indicates that the chronic hemiplegia is due not to hemorrhage or softening in some of the usual sites of such lesions, but to

tumor, abscess, meningitis, or diffuse syphilitic disease. General convulsions in hemiplegics may be due to focal lesions, the spasm, at first local, diffusing so rapidly that its probable focal nature is not recognized; in other cases they may be due to dural irritation, or to toxic causes, as when the hemiplegic suffers either from uremia or diabetes or is the victim of some intercurrent intoxication or infection. While, therefore, either Jacksonian epilepsy or general convulsions may occur in any of the more usual forms of hemiplegia or monoplegia, their presence should lead to careful examinations for the symptoms of other affections like those enumerated.

Prognosis.—The prognosis of chronic hemiplegia cannot well be generalized, owing to the great difference in the individual cases. A certain limit of improvement is soon reached in cases due to focal destructive lesions, such as hemorrhage and softening, but these will sometimes improve for a year or more. If removed from all sources of physical and mental strain, hemiplegics may live for years. Many of the hospital cases survive from ten to fifteen years and even much longer. The age and general health of the patient are important elements in the prognosis as to length of life. The hemiplegic syndrome is more serious in the old, in the very young, and in those suffering from diseases which cause constitutional infirmity. Apoplectic attacks are occasionally repeated, adding to the gravity of the prognosis. The condition of the heart, kidneys, and vessels is of especial prognostic importance. These remarks apply to the prognosis of chronic hemiplegia of the most common types. When the paralysis is due to tumor, diffuse syphilis, intoxication, infection, or hysteria, the prognosis is more favorable; but these diseases do not require to be especially considered in this connection, nor are we here concerned with the prognosis of cases associated with degenerative diseases or with the so-called neuroses.

Treatment.—*Prophylaxis and Internal Medication.* The prophylaxis of hemiplegia is, of course, usually that of the apoplexies from which it originates; but, as patients rarely expect such attacks, little, as a rule, is done for their prevention. As the hemiplegic, however, is often anxious about second attacks, it is not so difficult to have him carry out rules of careful living that may be of value in warding them off. Whenever there is good reason to apprehend the occurrence of hemorrhagic apoplexy, those threatened should lead most careful lives. In the majority of cases chronic degeneration of the arteries, associated with cardiac and renal disease, is present, and sometimes there may be a syphilitic or a gouty disease of the vessels. Strict attention should therefore be paid to the kidneys, heart, and vessels. Mild diuretics, especially the alkaline mineral waters, can be used in moderation. Constipation should not be permitted. The well known pills of aloin, strychnine, and belladonna, with an occasional mercurial and saline, are of service. If hypertrophy of the

heart or some other form of cardiac disease be present, a judicious use of cardiac remedies may be instrumental in preventing the apoplexy. Arsenic and small doses of the iodides are of some value, if long continued, because of their effects upon the vessels and viscera. The food should be sufficient in amount, but of such a character as to be easily digested and assimilated. Alcohol should not be abused, and in most cases it is well to abstain entirely from its use. Overwork, worry, and exposure to sudden alternations of heat and cold may act as predisposing as well as exciting causes of an apoplectic attack. While violent exercise should be carefully avoided, the hemiplegic should not lead a life that is too sedentary. All sources of physical and mental strain should as far as possible be removed, and exposure to excesses of temperature should be avoided. If patients have a syphilitic history they should be treated for chronic syphilis. Sodium and potassium iodide are valuable for their effects on the vessels and viscera. Lithium and colchicum preparations should be used in gouty cases. Occasionally it may be possible to prevent a hemiplegic attack, as when a syphilitic patient is troubled with numbness and formications or with slight spasms and twitchings, when active specific treatment may prevent the threatened attack. For most cases of hemiplegia and monoplegia of organic origin little can be done to affect the lesion responsible for the paralysis and the accompanying conditions. In cases of hemorrhage some absorption takes place as time progresses, and, in the hope of assisting this process, ammonium preparations and iodides may be used in moderation for a few weeks or months after the patient has rallied from the apoplectic attack. These drugs do some good also by their effect on the chronic disease of the vessels which is so commonly present. Necrosed tissues cannot be removed nor improved by drugs, so that in cases following embolism or thrombosis alteratives and absorbents are of value only for their effects upon the vessels. If a hemiplegia is found to be due to a tumor or diffuse syphilis, or to an intoxication, the patient should be treated accordingly. Close attention should be paid by the physician to the sufferings of hemiplegics. The pains in their limbs may be due to neuritis, and this should be treated by rest, hot applications, and internal remedies, like the salicylates, iodides, and bromides. Counterirritation and anodyne liniments may afford some relief.

Electrical Treatment. The question of the use of electricity for the treatment of organic hemiplegias is one of considerable interest. Some writers, like Hirt, seem to give it too much importance; others pass it by with neglect or even contempt. By its careful and judicious use a certain amount of good can be accomplished. The lesion cannot be removed by it, nor can secondary degenerations be prevented or benefited. It may, however, stimulate a palsied limb to do all that it is capable of performing, the amount of paralysis

present being sometimes out of proportion to the lesion causing it. The patient is paralyzed not only because of the destructive cerebral lesion which is present, but because of disuse, indifference, or psychical impression. Electrical stimulation leads the patient to efforts which he might otherwise neglect. Some of the incidents and accidents of hemiplegia may be benefited by electricity. It may be of service, for example, in the treatment of the secondary neuritis to which reference has been made, especially when it has become subacute and chronic; or it may promote absorption in the joints or improve the circulation and nutrition. The faradic current answers for most purposes in the treatment of the paralysis itself; but for chronic neuritis and arthropathies the galvanic current of moderate strength is more useful. Strong currents carelessly applied to paralyzed limbs may do harm, not only by undue stimulation of nerves and muscles, but occasionally by reflexly exciting the nervous centres. Electricity should not be used at all after an apoplectic attack for at least four weeks, and it may be wise to wait even longer than this. Treatment should not be begun as long as any evidences of brain irritation are present. As regards the treatment of contractures, in my experience, neither the application of galvanism to the muscles spasmodically affected, nor that of faradism or galvanism to their antagonists, has ever done any permanent good.

Massage and other Forms of Local Treatment. Massage, Swedish movements, and systematized exercises have some place in the treatment of residual hemiplegias; but too much should not be expected from them, and patients should not be turned over wholly to masseurs and masseuses, as they often believe that they can accomplish more than is possible. Massage and movement treatment, if violent, may be injurious rather than beneficial, but carried out with discretion and under the advice and control of the physician they may be of considerable service. It is sometimes a comfort to old hemiplegics with marked contractures to have them temporarily overcome, and patients may be seen straightening their fingers and hands with the unaffected hands. For the purpose of giving such temporary relief, splints may be carefully applied to the upper extremity, a small cylinder or rubber ball being placed in the hand; or instead of this an Esmarch bandage may be applied for a short time. Gymnastic exercises for the fingers and hand, as on a typewriter or a dumb piano keyboard, have been recommended for the athetoid and ataxic movements; but little can be expected from their use. The non-paralyzed as well as the paralyzed side of the body should receive local treatment, for, as has been shown, this side is often considerably affected, and the loss of power and of coordination may be improved by skilful and well regulated passive and active exercises. Surgical treatment for the contractures is sometimes practised, but, as a rule, should not be undertaken.

HYPERTROPHY OF THE BRAIN.

Some of the malformations of the brain and its envelopes have been considered in Chapter III. (pages 313 to 320), and, as there stated, most of these have but little clinical interest, while some, such as microcephalus, will be more conveniently discussed under idiocy and insanity. Before considering the cerebral palsies of children, however, a few of the more important cerebral defects, such as hypertrophy of the brain, heterotopia, atrophy of the brain, and porencephaly, should receive attention, as they are in some instances the pathological causes of these affections. Hypertrophy of the brain, or *macrencephalia*, is an enlargement or overgrowth of the whole or of a portion of the brain, although the partial hypertrophies usually receive special designations. It is to be understood that the increase in bulk is not due to abscess, tumor, or other acquired lesions, focal or diffuse. The condition is usually of congenital origin. It may give rise to *macrocephalus*, or enlargement of the head, but it may be present, for a time at least, without increase in the general bulk of the head, and macrocephalus may be due to hydrocephalus and other causes besides hypertrophy of the brain. In a strict sense hypertrophy refers to overnourishment, and the word *auxesis*, which is derived from the Greek *αὔξω* and means increase in size or bulk, would be more correct for these cases. Partial hypertrophy of the brain is a very rare affection. A few well authenticated cases of general enlargement of the brain with increase in the size of the head have been reported. Many years since, with Dr. F. X. Dercum, I assisted in making an autopsy on a child who had been supposed to have hydrocephalus, in which the brain was generally hypertrophied. In a case reported by Tuke, the patient having attained adult age, the right hemisphere of the cerebrum was very much enlarged, was tough, and weighed only twenty-three and a half ounces. The specific gravity of the two hemispheres was the same. The basal ganglia did not appear to have participated in the enlargement.

Symptomatology.—The principal symptoms in cases of congenital infantile hypertrophy are headache, tinnitus, apathy, at times great excitement followed by coma, mental deficiency, difficulty in walking, and convulsions. In eight cases under Beach's care, four died of convulsions, two in a comatose condition, and the remaining two of diarrhea and bronchitis. Idiotic and imbecile children suffering from hypertrophy of the brain usually die between the ages of five and fifteen years. Down gives the following description of the peculiarities presented by one of these cases: "He walks circumspectly, and when in the act of prehension does it in a slow deliberate manner; even the temptation of taking food to his mouth (and he is particularly fond of eating) does not induce him to feed himself in other than the most deliberate way. He talks, but it is in the same

slow manner, and only in response to questions. His sentences are short and their utterance tardy. Even when excited by promise of reward he speaks with only slightly increased utterance. His progression is by slow walking, and no inducement makes him run. He is rather below the ordinary stature; has no deformity, except that his fingers are shorter than normal. His face is largely developed, and his nose correspondingly so. When questioned he frequently makes feeble movements of the lips without utterance of sound, and only gives audible responses when stimulated to do so. He is seventeen years of age, and the signs of puberty are not wanting." When the head is very much increased in size, the patient is sometimes unable to carry it without support.

Etiology and Pathology.—The causes are obscure, but probably tuberculous, syphilitic, alcoholic, or other degenerative heredity is more efficient than anything else. Andral and some early observers have attributed hypertrophy to lead poison, but the proofs of this relationship are, to say the least, uncertain. According to Virchow, who wrote on this subject as early as 1856, the increase in the bulk of the brain is due to overgrowth of the neuroglia; and most subsequent observers agree with this view. Rokitansky believed that increase in size was probably due to an albuminoid infiltration of the brain structure. According to Fletcher Beach, the disease as seen in imbeciles is due to what appears to be granular matter; but this appearance may be dependent upon the increased amount of connective tissue which has broken down as the result of postmortem changes. The brain is anemic, and the bloodvessels are attenuated, but in rare cases they are increased, and a large number of leucocytes may be present. It has been suggested that the anemia which is so constant may occur only during the last stages of life, as the result of the final compression of the brain within the skull. The skull cap may be thinned or thickened. The dura is adherent to the cranium, and the convolutions are flattened and pressed together. The white matter is usually in excess. Cerebrospinal fluid is deficient or absolutely lacking in the membranous spaces, and absent or in small quantities in the ventricles. Some cases have been recorded in which local enlargements have been present in the peripheral nerves, in the ganglia, and in the central nervous system. A tendency to enlargement of the head unassociated with hydrocephalus has been observed in several generations. The consistency of the brain is nearly always markedly changed. It has been described as tough like the boiled white of an egg, or like cheese or rubber. Increase in the bulk and weight of the brain is sometimes very great. In one reported case it reached nineteen hundred and eleven grammes, or 65.7 ounces. (Virchow.) This brain is one of the heaviest on record, but several cases have been reported in which the weight has reached from sixteen hundred to nineteen hundred grammes.

The following table is slightly modified from one by Beach:

Weight in Ounces of Seven Hypertrophied Brains, and Average Weight of Brains of Individuals of the Same Age.

AGE.	WEIGHT OF HYPERTROPHIED BRAINS.	AVERAGE WEIGHT OF BRAINS (DR. BOYD'S TABLES).	AVERAGE WEIGHT OF BRAINS (DR. BEACH'S TABLES).
5	49.5	40.23	40.5
8	53	45.96	39
10	55	45.96	40
11	49	45.96	41
11	49.5	45.96	41
14	52	45.96	41
15	62	48.54	42

Diagnosis, Prognosis, and Treatment.—The diagnosis is chiefly to be made from chronic hydrocephalus, this resting on the history of the case and a careful examination of the head. The following table shows the points of difference as given by Beach:

HYPERTROPHY OF THE BRAIN.	HYDROCEPHALUS.
Head increased in size, but not so much as in hydrocephalus.	The head more increased in size.
Increase is most marked above the superciliary ridges.	The size of the head most marked at the temples.
The head approaches a square shape.	Head more globular.
No elasticity over the fontanelles.	Elasticity over the closed fontanelles.
Often depressions in the position of the fontanelles.	No depressions in the position of the fontanelles.
Distance between the eyes not increased.	Distance between the eyes increased.

The prognosis is of course bad, and, so far as the affection is known, no treatment is available.

HETEROTOPIA.

The term heterotopia, as used in neurology, is applied to the misplacement or the development in unusual positions of either the gray or the white matter of the brain and spinal cord. Numerous cases of heterotopia of the brain have been recorded. Although a true spinal heterotopia is sometimes found, Van Gieson has shown that many cases of so-called heterotopia of the cord may be due to post-mortem manipulations. Heterotopia is to be classed as a peculiar developmental aberration rather than as a special hypertrophy. It has little clinical importance. The observed cases have been found in connection with idiocy and imbecility. Cerebellar heterotopia has been discovered after death, the individual not having shown abnormal manifestations during life. Cerebral heterotopia is almost invariably associated with mental or physical arrest, and is usually a part of some more general congenital morbid condition. It has been found in the brains of those suffering from some forms of infantile

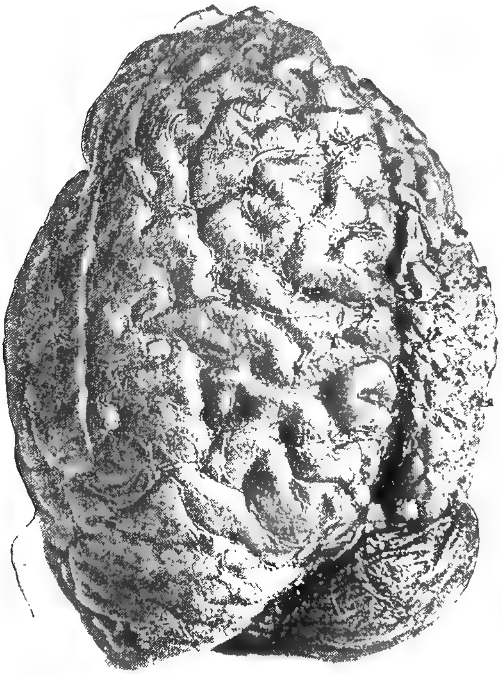
cerebral palsy with idiocy and epilepsy. The isolated masses of cinerea, when cerebral, are commonly found near the basal ganglia or the walls of the ventricles, especially of the lateral ventricles. They are also somewhat frequently located in the white matter of the cerebellum. They have practically the same structure as the cerebral cortex, from which they are regarded as having been detached. Matell has reported the case of a woman, a low grade imbecile, who died at the age of twenty-five years, and who had suffered since her sixth year from epilepsy. The patient's head was small, and showed abnormalities. The forebrain was small, and its fissuration reduced and aberrant. A strip of gray matter was present in the centrum ovale. The area in structure was like that of the cortical substance, with which it was connected here and there by bridge-like formations of cinerea. Matell regarded the anomaly as dependent either on an interruption of the development of the brain, or on a complete standstill of its growth at an early period, as at the sixth month of fetal life. The condition was present in both hemispheres. This case is especially referred to here because the investigation seemed clearly to show its cortical origin. In other cases the insular masses are so far removed from the cortex as apparently to have no connection with it, and yet these, like the striatum itself, are from an embryonal standpoint probably cortical structures. It is a matter of dispute among anatomists and morphologists whether the heterotopia is due to aberrant or abnormal development of nerve fibres causing detachment of cortical or ganglionic cinerea, or whether the gray masses develop primarily in unusual positions and are subsequently entrapped by the fibres. Heterotopia of the white matter has been observed in the oblongata. Heard, in 1894, collected the observations of this kind, which, including his own, amounted to nine in all. In the majority of these cases anomalous bundles of white matter have been found unilaterally situated. One was observed by Cramer upon the side of the lesion in a case of cerebellar hemiatrophy. Kronthal discovered two anomalous unilateral bundles in a case of bulbar paralysis. Heard concludes that the most frequent and fairly constant anomaly in these cases appears to be the presence of a column of white nerve fibres arising at the level of the decussation and thence proceeding cephalad in a position internal to the substantia gelatinosa. Klob found a mass of white cerebral substance the size of a bean hanging from a pedicle between the two optic nerves.

ATROPHY OF THE BRAIN—ATROPHY AND SCLEROSIS.

Anencephaly, or absence of the brain, when total, is without clinical interest, but certain forms of atrophy of the brain give rise to partial anencephaly and to affections which may call for diagnosis and in a few cases for treatment. The more generalized forms of

encephalic atrophy are usually congenital. Partial or diffuse atrophy may be congenital, may be initiated at the time of birth, or may be acquired after birth. When postnatal it commonly originates in the very earliest years of life. In partial anencephaly due to atrophy both hemispheres may be in part wanting, or one may be present and the other practically absent, or one or both hemispheres may be the seat of defects of varying size. Porencephaly is in a general sense a form of partial anencephaly, but in the cases to which reference is made in this section, although a large portion of the brain may be

FIG. 315.

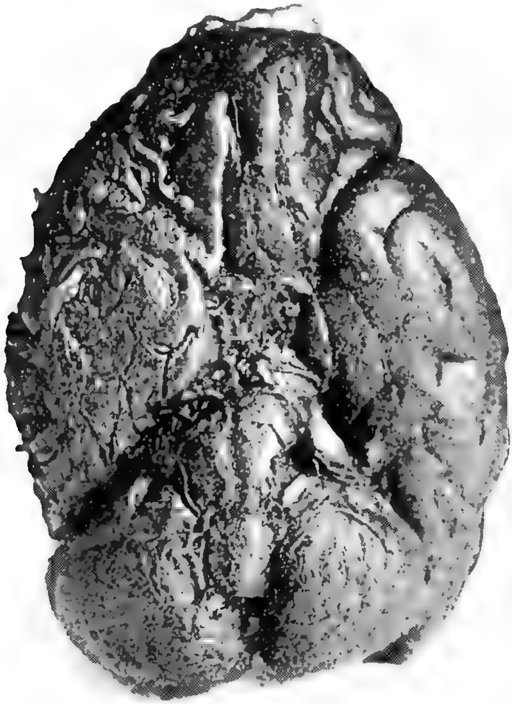


Coronal aspect of the brain, showing marked atrophy of the right hemisphere and of its convolutions, from a girl of nineteen, with left hemiplegia, moderate contractures in the paralyzed limbs, epilepsy, and imbecility. (Shaw.)

absent, a circumscribed hole or cavity is not formed. In general atrophy brain and skull are both small. When any one considerable portion of the brain is absent, other functionally associated regions are also found to be more or less atrophied or involuted, as when atrophy of one cerebral hemisphere is associated with marked diminution in bulk of the opposite olive and opposite lateral lobe of the cerebellum. Occasionally the entire cerebellum or a very large portion of it is atrophied or undeveloped. Reference was made in Chapter IV. to these cerebellar cases, and to the absence or atrophy of such important structures as the callosum.

Etiology and Pathology.—Among the causes of partial congenital atrophy of the brain are pathological conditions of the amnion, as adhesions of this membrane to the embryo, or constrictions of the cephalic extremity of the embryo. Such adhesions and constrictions may be caused by pressure either from within or from without, and they occasionally result from abdominal traumatisms received by the mother. Acquired atrophy may be due to meningeal hemorrhage, to meningitis or encephalitis, or to any cause injuriously

FIG. 316.



Basal aspect of the brain shown in Fig. 315: extreme atrophy of the right hemisphere, decided atrophy of the right half of the pons, and small left lateral lobe of the cerebellum. (Shaw.)

affecting the nutrition of the infantile cortex. In rare cases the atrophied brain is less consistent than normal, but usually it is firmer and is made up largely of connective tissue. In other words, atrophy and sclerosis are commonly associated. Osler found wasting and induration in fifty cases. This was either in groups of convolutions or involved an entire lobe or in some cases the whole hemisphere. The sclerosis in most of these cases is secondary to atrophy of the true nerve elements. When sclerosis is the primary lesion a diminution in bulk is not likely to be present. The causes already detailed, and others, may lead to an arrest of development of the

nerve elements, and consequent overgrowth of connective tissue. Under various names, such as *lobar sclerosis, tuberous sclerosis,* and *diffuse sclerosis,* much attention has been paid to this subject, especially by French observers in recent years, prominent among them being Bourneville and Cotard. Now and then, instead of being lobar and diffuse, the sclerosis may be in nodules or patches, and these may be scattered over the surface of the brain. "The affected convolutions are small, of a grayish yellow color, often with a stippled, pitted surface, to which the pia adheres firmly. In contrast with the neighboring normal gyri, the appearance is very striking and

FIG. 317.

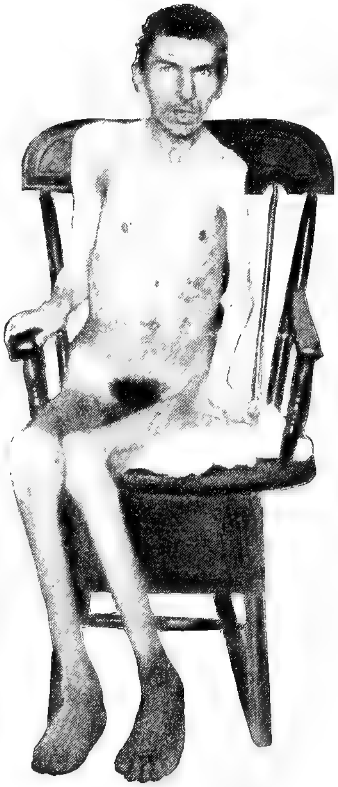
Coronal aspect of the brain in a case of extreme atrophy of the right hemicerebrum. (Shaw.)

characteristic. The reduction in size of the affected hemisphere may amount to one third of the bulk. In one case the atrophied hemisphere weighed two hundred and sixty grains, the normal ten hundred and seven grains. The tissue may be a mere shell over a dilated lateral ventricle, as in the cases of Baud and Piorry. In many cases anfractuosities and small cysts have been found in and about the sclerosed tissue." (Osler.) In Figs. 315 and 316 are shown the appearances presented by the superior and inferior aspects of the brain from a case of atrophy reported by Shaw. The right lateral lobe of the cerebellum is exposed; and, as shown in Fig. 316, the right half of the pons and the left cerebellar lobe are decidedly reduced in size. The brain was taken from a girl of nineteen, who was imbecile and epileptic and had marked left hemiplegia

with moderate contractures in the paralyzed limb. In Fig. 317 is shown the brain from another remarkable case of cerebral atrophy reported by Shaw. The right hemicerebrum is extremely atrophied, so that both the cerebellum and the quadrigeminal body are uncovered. The patient from whom the specimen was taken was an imbecile with paralysis and extreme atrophy of the trunk and limbs, and was subject to convulsions. (Fig. 318.) The diagnosis, prognosis, and treatment of atrophy of the brain, general or partial, are considered under imbecility, infantile cerebral paralysis, and other affections of which it is the pathological cause.

Symptomatology.—The symptoms of atrophy of the brain will vary according as it is general or partial, and to some extent according to its location. Almost invariably mental defect is present, and this is not infrequently a low grade of idiocy. Some of the forms of cerebral palsy in children are associated with unilateral and bilateral atrophies.

FIG. 318.



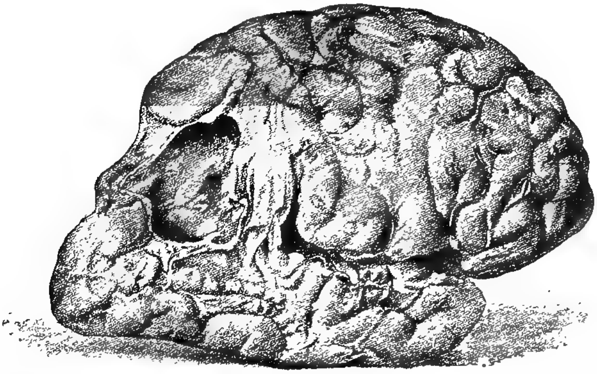
Infantile cerebral hemiplegia, with marked atrophy of the limbs and trunk; the patient imbecile and epileptic. The brain from this case is shown in Fig. 317. (Shaw.)

PORENCEPHALY.

The term porencephaly is derived from the Greek words πόρος, a cavity, and εγκέφαλος, brain, meaning a hole or cavity in the brain, and has been used in describing somewhat different conditions. Heschl, who suggested the name, applied it only to congenital cases, the defect, as described by him, being one in which, because of an absence of cortical and subcortical substance, a large hole, usually somewhat funnel-shaped, was found. This cavity in some instances communicates with the ventricles. The term is applied by some authors to acquired as well as to congenital cases; others would restrict it to the latter. Besides the many cases which are clearly traceable to the fetal period, a few have been recorded in which cavities of considerable size have originated after birth, some of them as the result of meningeal hemorrhage, or from intracerebral hemorrhage, embolism, or thrombosis. As Sachs has suggested, some of the so-called congenital

cases may be regarded as acquired in the sense that they have resulted from influences acting on the fetal brain. Cases acquired after birth are sometimes spoken of as spurious porencephaly. The mechanism of the production of porencephaly is not entirely clear. Probably in cases of hemorrhagic origin the effused blood by compression causes atrophy and arrest of development in the cortex and subcortex; and when vessels are occluded a necrosis of the tissues results, with some additional arrest of development in adjacent regions. The blood is absorbed in hemorrhagic cases, and the cavity formed by the loss of brain substance is filled with cerebrospinal fluid. Cases of acquired porencephalus have been reported by Peterson, Willard and Lloyd, and others. Certain characteristics seem to belong to all the cases of porencephaly of congenital origin. When the defect is intracerebral the overhanging cortex is depressed and roofed in by the arach-

FIG. 319.

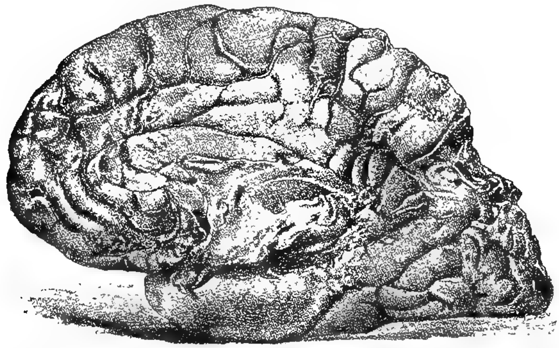


Lateral aspect of the right hemicerebrum from a case of porencephaly in a man fifty-seven years of age: the patient's body observed to be unsymmetrical at birth; arrested development of the left side; asymmetry of the skull; history of a fall and of intemperance and sunstroke; absent knee jerk and unequal pupils; general paresis late in the history of the case. (Brush.)

noid, the pia closely investing the convolutions. When the subarachnoid space forms a part of the cavity or communicates with it, the arachnoid terminates along the edges of the cavity, while the pia invests the brain for some distance into the cavity. According to Kundrat, one of the most characteristic features of the congenital cases is a peculiar radiating arrangement of the fissures and convolutions around the cavity. An important point bearing on the time of initiation of the porencephalic lesion is the fact that the permanent primary fissures are never entirely absent, although they may be considerably changed in extent and direction. Porencephaly attacks both hemispheres in about one third of the cases. When only one side of the brain is affected, this is somewhat more frequently the right than the left hemisphere. The convexity of the parietal lobe is its most frequent seat, but it has been found in all parts of the

brain. According to Brill, no case has been reported in which the mesal surface of the hemisphere has been implicated; but, in contradiction to this, a case reported by Brush (Figs. 319 and 320) shows distinct involvement of the mesal surface. The cavity, it will be observed, occupies the position of the parietal lobe, and encroaches upon the occipital and temporal lobes. On the inner surface of the hemisphere the cuneus is represented by a shell of brain substance, and nothing remains of the quadrate lobule but a thin stratum of brain substance at its anterior portion. The symptoms of both congenital and acquired porencephaly are those which are described as belonging to some forms of diplegia and hemiplegia. Mental disorder is always prominent. Various grades of idiocy are present; this may be of such an extreme character that the patient leads little more than a vegetative life. Mutism is sometimes absolute, and, as a rule, speech is greatly impaired; deafness and in rare instances blindness are present. Strabismus is common.

FIG. 320.



Mesal aspect of the right hemicerebrum in a case of porencephaly. Same case as Fig. 319. (Brush.)

Epilepsy occurs in a fair percentage of cases. The most constant manifestations besides the mental deficiency are, however, in the trophic and motor spheres. Extreme conditions of spastic paralysis with contractures, athetoid and choreoid movements, and atrophy and malformations of the limbs, trunk, and head, are frequently present. While the above statements summarize the symptomatology of the vast majority of cases of porencephaly, this defect may exist and be of considerable size without extreme conditions of paralysis and imbecility. The diagnosis of porencephaly is in the main that of the infantile diplegias and hemiplegias, which will be considered in the next section. It is occasionally important to differentiate porencephaly from tumor, meningitis, or other more active lesion, as the former is less amenable to treatment. The prognosis of porencephalic cases is necessarily bad, although many of them reach adult life.

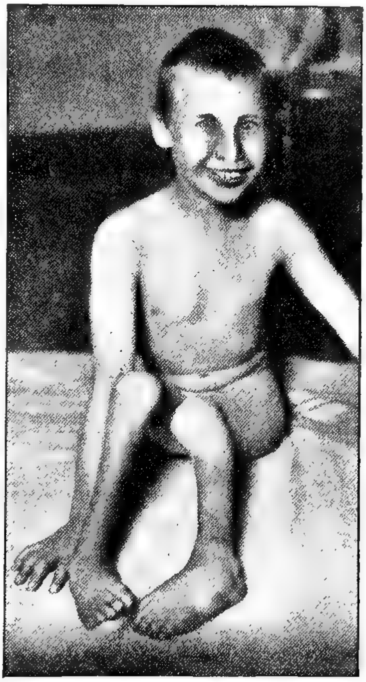
THE CEREBRAL PARALYSES OF CHILDREN, AND AFFECTIONS ALLIED IN PATHOLOGICAL CAUSATION.

Under the general designation "cerebral paralyses of children" or "infantile cerebral palsies" are included several affections differing somewhat in their clinical features, but agreeing in that their symptoms are due to lesions which involve chiefly the corticospinal portion of the motor subdivision of the nervous system. While paralysis, rigidity, and other phenomena are the predominating features, other manifestations, such as defective intelligence often amounting to imbecility or even idiocy, disturbances of the special senses, and vasomotor and trophic disorders, are frequently present. Paralysis may indeed be absent, and yet the case may pathologically belong to the general type classed as cerebral palsies, the lesions being so situated as not to destroy those parts of the brain which are related to motor functions. The literature of the subject has advanced with rapid strides during the last ten years. For its full discussion special monographs should be consulted, among the most important of which are contributions by Cotard, Kundrat, Strümpell, Marie, Ross, McNutt, Knapp, Osler, Freud, Rosenthal, and Sachs and Peterson, although many others almost equally valuable have been published. An important pioneer communication was by Little, in 1853, on the spastic paralyses and rigidities occurring in children, this writer first clearly relating some of these affections to abnormal parturition, difficult labors, and premature birth. Dr. Sarah McNutt was the first to show the relations of meningeal hemorrhages to these affections. As early as 1879 the author, in a lecture published in the New York *Medical Record*, reported several cases of infantile diplegia and hemiplegia, discussing the question of their cerebral origin. Gee had previously reported similar cases. Freud and Rie and Rosenthal have recently done much towards systematizing and making more complete our knowledge of the diplegias. The cases are usually considered under the heads of *diplegias*, *paraplegias*, *hemiplegias*, and *monoplegias*. These terms, embodying the idea of paralysis, are strictly applicable to a majority of cases, but are not descriptive of all. The motor phenomena are frequently of the nature of rigidities, choreas, athetoses, ataxias, or other affections characterized by increased muscular activity, and these may be associated with paresis and paralysis, but in other instances real loss of power is absent. They are *hyperkineses*, disorders in which motility is exaggerated, rather than *akineses*, or paralyses, in which motion is impaired or lost. In the present state of our knowledge, however, little would be gained by an attempt to classify the hyperkineses apart from the paralyses. Hemiplegias constitute a large majority of the whole number of cases, although diplegias are by no means rare. An analysis of two hundred and twenty-five cases made by Sachs and

Peterson shows eighty-one cases of right hemiplegia, seventy-five of left hemiplegia, thirty-nine of diplegia, and thirty of paraplegia. Pure monoplegias are very rare.

Varieties of Diplegia.—The cerebral diplegias exhibit considerable clinical diversity in their symptomatology, but a fair practical basis for the study of these affections is afforded by the four types suggested by Freud, namely, (1) generalized rigidity; (2) paraplegic rigidity; (3) double spasmodic hemiplegia; and (4) generalized bilateral chorea and double athetosis. These designations aptly describe the dominating features in each set of cases. Generalized rigidity is sometimes spoken of as Little's disease. A more or less rigid condition is presented by the muscles of the trunk and extremities, and in some cases even by those of the neck and face. In the typical paraplegic cases rigidity is confined to the lower extremities. (Fig. 321.) In both of these varieties true paralysis is not a marked feature, or may be absent. When rigidity and contractures are general and are of a decided character, it is sometimes difficult to determine how much real paralysis is present. In the double spasmodic hemiplegias true paralysis is clearly associated with the spastic state, and not infrequently the limbs exhibit more or less atrophy. Instead of typical cases of generalized rigidity or double spasmodic hemiplegia, my own investigations have shown that a condition of rigidity or a spasmodic hemiplegia is often present on one side while on the other side one extremity only may be markedly affected, this being usually the leg. (Figs. 322 and 323.) While the arm may escape in large part, however, examination will usually show some impairment of power and some hypertonia. In cases of generalized bilateral chorea and double athetosis the hyperkinesia is usually most marked in the upper half of the body, the movements often affecting the upper extremities, trunk, head, and face. These so-called types are often irregular and are seen shading into one another. Great differences in the degree of impairment of intelligence may be present, the relative amounts of rigidity and paralysis may vary considerably, and the prominent features of any one variety

FIG. 321.



Infantile spastic paraplegia. (Willard.)

may be exhibited in some degree by any other. Paraplegic rigidity is of course present in generalized rigidity, and chorea and athetosis often have some place in other types than the fourth. In Little's disease the rigidity may be extreme, moderate, or slight, or practically almost latent, some cases approaching closely to the normal. Strabismus, convulsions, or a greater or less degree of paralysis may be present in any form. Between the types of generalized rigidity and of paraplegic rigidity occur transitional forms. In the paraplegic cases, for example, traces of rigidity may be present in the upper limbs, such patients showing feeble contractures, slight involuntary movements, and a little awkwardness in using their hands. Between generalized rigidity and double spasmodic hemiplegia are also transitional forms. It may be interesting to record in this connection the chief features of two diplegic cases reported by me in 1879. Summarized, they were as follows: dilated pupils and internal strabismus; weakened intelligence; in one case convulsions in infancy; apparent weakness of the muscles of the back in both cases; marked spasticity of the lower extremities, chiefly affecting the flexors and adductors; a similar spastic condition in the arms in one case, and a tendency to spasmodic contraction in the other; inability to stand or walk alone; tendon reflexes increased; no paralysis of the bladder or bowels; retention of electrocontractility and sensibility; and absence of vasomotor changes or joint troubles.

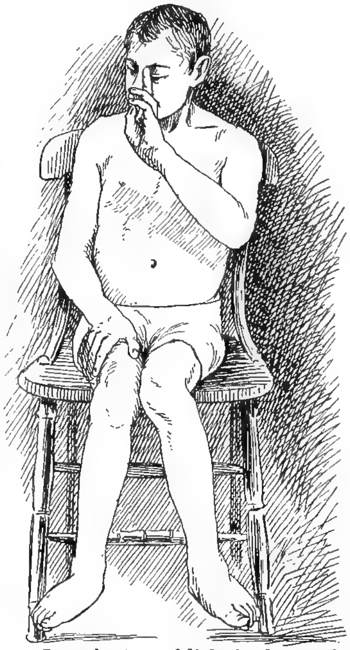
FIG. 322.

Idiocy with spastic diplegia; chiefly affecting both legs and the right arm; with rachitic deformities.

Varieties of Hemiplegia.—Many of the cases of infantile cerebral hemiplegia differ from each other in especial but easily determined particulars. Hemiplegia with spasticity and marked contracture is common; but instances of flaccid paralysis are met with, although they are rare. The hemiplegia may be associated with athetosis or athetoid movements, with chorea or choreiform movements, or with coarse tremors or tremulous movements of special character; or it may be combined with general arrest of development of the same half of the body, or with local arrest in a limb or limbs, as when confined to the distal portion of the arm. Instead of atrophy more or less diffuse hypertrophy may in rare

instances be present. Two of these types of hemiplegia are shown in Figs. 324 and 325. In other cases it may be associated with a motor or sensory aphasia, with hemianopsia, or with mirror writing. In a few cases the associated mental state is normal or nearly normal; in others the condition is one of feeble-mindedness or retarded intellectual development; and in still others, which constitute the largest number, true idiocy or imbecility is present. As in adults, the arm is more seriously and more frequently affected than the leg, and the paralysis is more marked in the distal than in the proximal portions of the limb. Compared with adult hemiplegias, motor aphasia occurs relatively oftener with left-sided paralysis, disorders of movement are more frequent, and arrest of development on the paralyzed side is decidedly more prominent. The face, and especially the lower face, may be involved, but it often escapes. This involvement of the face is much more common as a persistent phenomenon in the diplegias, and especially in the double spasmodic hemiplegias and in the bilateral choreas and double athetoses. The facial muscles in the latter cases are sometimes the seat of bizarre grimaces. One side of the face may show paresis and the other side contractures.

Fig. 323.



Irregular type of diplegia: low grade imbecile; unable to walk or talk; paraplegic rigidity; considerable rigidity of the shoulders, but arms freely movable below the elbows; athetoid movements in the upper extremities.

Symptoms of the Period of Onset.—The published accounts of the clinical history of cases of infantile and juvenile cerebral paralyses have, for manifest reasons, been based chiefly upon a study of the residual symptoms. A fetal symptomatology is unknown, as intra-uterine manifestations can be surmised only through the complaints of the mother. Cases which originate during labor or very early in infancy are frequently not recognized immediately as instances either of mental defect or of paralysis. In the so-called birth palsies, usually after a prolonged or difficult first labor, the child may be born asphyxiated, or may have a convulsion or a series of convulsions immediately or a short time after birth. While often present, asphyxia or convulsions are not invariable. The infant may exhibit simply a condition of extreme feebleness. When the causes have acted before birth, symptoms like coma or convulsions may call attention to the child at the time of birth. Labor may not

have been especially difficult. The appearance of the child may be such as to attract attention or to arouse suspicion; but it may be difficult or almost impossible to judge of the existence of mental arrest or of paralysis from the conditions present at or soon after birth. In a short time it may be noticed that the limbs are not used with the ordinary freedom, and that they tend to remain flexed or in unusual positions. General, paraplegic, or localized rigidity, grotesque facial expression, a tendency to keep the mouth open, drooling, and strabismus may be observed. Efforts are not made to walk or talk at the usual age; and the infant does not fix its attention or show purposive actions like normal children. As the hemiplegias as well as the diplegias acquired after birth are due to meningeal hemorrhage, intracerebral hemorrhage, embolism, thrombosis, focal encephalitis, acute or chronic meningitis, and hydrocephalus, the symptomatology of the onset must differ according to the causative affection. When the lesion is a meningeal hemorrhage the attacks are most frequently ushered in by loss of consciousness and convulsions. Coma is nearly always profound, and may vary in duration from a few hours to several days. Changes in temperature have been recorded and are probably usually present, but they have not yet been studied as carefully as in the apoplexies of adults. Delirium, vomiting, retraction or tossing of the head, or screaming spells may be present, and paralysis of one half of the body may in some cases be made out during the apoplectic attack. It is a point of interest that the hemiplegia may be of more complete character than in older patients. Unilateral disturbance of the respiratory muscles may be evident, and the paralysis is likely at first to be marked in the face as well as in the limbs. The reader is referred to Chapter V. for the discussion of the symptomatology and differential diagnosis of intracerebral hemorrhage, embolism, and thrombosis as they occur in adults, and to the various sections of Chapter III. for the symptoms of marantic and secondary thrombosis, of focal encephalitis, of the different varieties of acute or chronic meningitis, and of hydrocephalus.

Fig. 324.

Spastic hemiplegia with imbecility: marked arrest of development of the entire right side.

Symptoms and Conditions accompanying the Residual Paralysis.—After the symptoms of the acute attack have passed off, the residual symptoms presented by different cases, which have

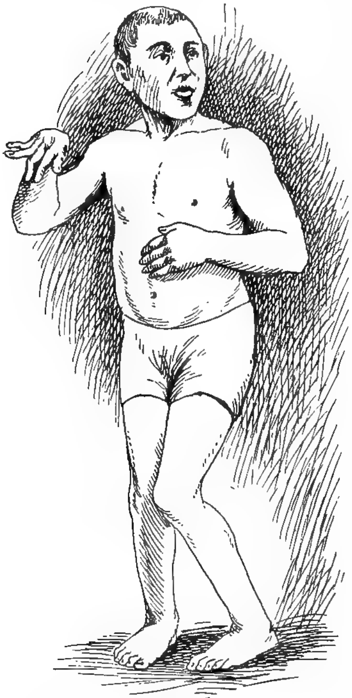
been enumerated under the varieties of diplegia and hemiplegia, will depend largely upon the position, extent, and character of the lesions which remain. In a few cases monosyllabic speech, nystagmus, dysphagia, a peculiar dyspnea, stridulous respiration, and arrest of laryngeal growth have been noted. As already stated, rigidity is the characteristic feature of two of the most important types of diplegia, but it may be present in other varieties, and also in the hemiplegias. It may be of any degree, from the lightest hypertonia which offers scarcely any obstacle to passive movements, to a rigidity which fixes the limb in an almost immovable position. Rigidity may be present with but little loss of power, or it may coincide with most pronounced paralysis. The difference between rigidity and paralysis is sometimes made apparent by the station and walk of the patients. When paralysis is present, the limbs give way and the patient may be unable to stand; when the condition is chiefly that of rigidity, both standing and walking may be possible, attitude and gait being of the spastic type. Contractures are probably present in three fourths of the cases of diplegia, and in a large but less percentage of cases of hemiplegia. Some of the characteristic attitudes and methods of progression are shown in Figs. 326 and 327. Adductions and flexions predominate, giving forms of the adductor or close-legged and of cross-legged progression. Equinus and equinovarus are among the most common deformities, but valgus is sometimes observed. Occasionally the deformities are due to secondary conditions in the tendons and joints rather than to spasms and contractures. The hyperkineses of hemiplegia and diplegia are sometimes spoken of as "postparaplegic" and "posthemiplegic" disorders of movement, and these terms are correct enough as applied to some cases; but the affections may be independent of any paralysis, as when we have choreiform or athetoid movements in the fourth of Freud's diplegic types. In other instances they are concomitant and

FIG. 325.

Spastic monoplegia dating from six years of age: convulsions from birth; atrophy and contractures of the left upper extremity; lower extremity but little affected.

associated phenomena rather than sequential to the paralyses. The general characteristics of these disorders of movement have been considered in the section on adult hemiplegias. Choreic and athetoid movements are not infrequently mingled in the same case, but examples of pure athetosis are occasionally observed. In Fig. 328 is an illustration of bilateral chorea and double athetosis, and in Figs. 326 and 329, of unilateral athetosis. Peterson has described a morbid movement to which he gives the name of posthemiplegic polymyoclonus. The movements consist of constant clonic contractions in the muscles of the limbs affected, not occurring synchronously, the rhythm being that of paralysis agitans. In one of the cases now in the Philadelphia Hospital a distinct athetosis is present in the fingers and hand, and at the same time the limb from the shoulder is in a constant state of slight movement, the arm being held downward in the position in which it is usually carried on standing or walking. In this case also slight twitchings are almost constantly present in the muscles about the mouth and the lower part of the face on both sides. In some cases the arm is twisted or distorted at the joints, and is carried around and held against the back, palm outward, to keep it at rest. Associated movements are frequently observed, especially among diplegics. Forced movements are less common, but forced attitudes which are compelled by the paralysis, spasm, and rigidity that are present, and it may be in some instances by the mental status of the patient, are frequent. Knee jerk and muscle jerk are usually exaggerated: and ankle clonus and front tap movements can often be elicited. If the knee jerk should be absent, the possibility of a spinal complication should be borne in mind. It may appear to be absent when it is not, the limb being simply held spasmodically fixed. Strabismus occurs with moderate frequency. The muscles in diplegia and hemiplegia respond to the electrical current. The parts affected, however, are arrested in growth. In the hemiplegic cases, especially in those which have lasted some

Fig. 326.



Irregular type of diplegia in a low grade demi-microcephalic idiot: both legs spastic; right leg smaller and weaker than the left; rigidity, paresis, and atrophy of right arm; left arm unaffected or but slightly involved; method of progression and athetoid movements on using the right arm shown.

years, not only are the limbs shorter and smaller, but the trunk and head and in fact the entire half of the body are distinctly diminished in size. This is evident in some of the illustrations. The unparalyzed as well as the paralyzed side may be retarded in growth. In extreme cases of diplegia the limbs sometimes present a wasted appearance; often they are of fair bulk and firm and hard. In choreoid and athetoid cases occasionally the muscles are hypertrophied. In the case shown in Fig. 328 the entire upper half of the body seems to be overdeveloped. In many cases of infantile cerebral palsy the cranium shows defects and asymmetries which are often attributed to the use of instruments in difficult labors, and in rare cases cranial deformities are thus produced. Not infrequently the skulls are smaller than normal, being demimicrocephalic. The skull may be too long, or too short, or oblique, or may present any of the peculiarities described in the section on symptomatology and methods of investigation. In the hemiplegic cases the skull is often notably flattened on the side opposite the paralysis. Other of the so-called stigmata of degeneration, such as abnormalities of the face or of the palate, teeth, ears, or hair, may be present. On page 149 is given a table which shows the comparative measurements of twenty cases of infantile spastic hemiplegia studied by Peterson and Fisher. They indicate marked deviations from the normal.

FIG. 327.

Diplegia showing cross-legged progression.

Epilepsy.—A few of the symptoms or associated conditions need special consideration. No convulsions were present in about one half the cases reported by Rosenthal. Sachs records epilepsy in forty-five per cent. of all his cases. All cases with a history of convulsions are not to be regarded as epileptic, as such convulsions may occur daily once or twice at or near the time of onset of the paralysis, and may be due to the immediate exciting effects of the lesion. Marie, Freud, Sachs, and others have directed attention to the important practical point that some of the cases regarded as examples of ordinary epilepsy will be found on close examination to belong properly to the diplegias or hemiplegias, only conditions

of slight paresis or rigidity being present and overlooked. Such cases are not benefited as much by bromide treatment as are epileptics of the more usual types. The spasms may be general or

FIG. 328.

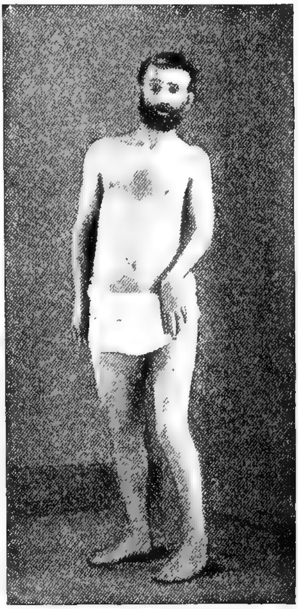
Bilateral chorea and double athetosis: face, trunk, and upper extremities chiefly affected; patient a deaf mute and mentally deficient; convulsions the third day after birth; walked at the age of nine years; movements said to have begun at about the age of twelve years; mother had a severe fall during ninth month of pregnancy. (Dercum.)

of the Jacksonian form, and, when the latter, may in time become more and more generalized. Sometimes the convulsions are re-

garded as a cause of the paralysis rather than as a result or even a concomitant; and, as Dercum suggests, it is conceivable that a toxemia may give rise to severe convulsions, and that these may in turn so affect the nutrition of the brain as to lead to paralysis.

Defects in the Visual Fields, and Mirror Writing.—Hemianopsia and sector or irregular defects in the visual field are occasionally observed in infantile hemiplegias; or such visual defects may be present in cases which should be included under the class of cerebral palsies although no motor loss may be present. The initial and residual lesions in these cases are outside of the motor areas and tracts. In the case reported by Brush, Figs. 319 and 320, such visual changes may have been present, as the cuneus was simply represented by a shell of brain substance. In one case studied by me at the Vineland Training School the patient, who was at the time sixteen years old, could read, write, and draw, had fair powers of memory and attention, but was nervous, excitable, and obstinate, and at irregular intervals had severe convulsions, in which the head and eyes were turned to the right and the face and limbs of the right side were in violent spasm both tonic and clonic. Before the convulsions he passed into a dreamy state, and on other occasions had visual hallucinations. He had hyperopic astigmatism of both eyes. Ophthalmoscopic examination by Dr. S. D. Risley showed woolly choroid and the general eyeground of a fluffy red color. The field in the right eye was contracted. In the left eye the nasal field was cut off abruptly in a vertical line three to five inches to the right of fixation point. Mirror writing is sometimes present in infantile cerebral hemiplegia. An interesting case at the Vineland Training School when eighteen months old had an illness attended with convulsions and resulting in partial right hemiplegia. The boy ranked with the highest grade of imbeciles. He could repeat the alphabet, and spell and read words of one syllable. Right sided partial paralysis with atrophy was present, as shown in Fig. 330. When efforts were made to teach him to write with his left hand from a copy it was discovered that he always made his attempts at writing from left to right. He copied slowly, but always began without hesitation to write in mirror fashion.

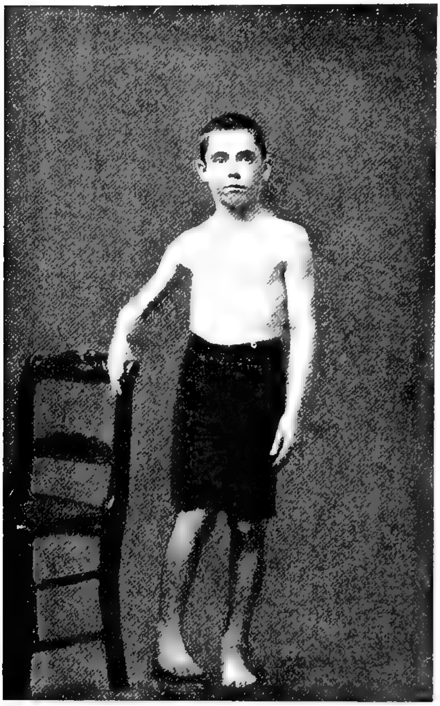
Fig. 329.



Hemiplegia with atrophy and athetosis in a patient forty-four years old; the paralysis dating from early childhood. Marked depression in the right frontoparietal region.

Etiology and Pathogenesis.—The etiology of a certain number of cases of infantile cerebral palsies can only be surmised. The causes have been traced in a larger percentage of diplegias and paraplegias than of hemiplegias, one reason for this being that traumatism during labor is a more frequent cause of the former than of the latter. Both diplegias and hemiplegias can be classified from the etiological standpoint into cases which originate during the intra-uterine period, cases due to traumatism during labor, and cases due to various causes acting after birth.

Fig. 330.



Infantile cerebral hemiplegia with feeble-mindedness; the patient being a mirror writer.

Prenatal Causes. With regard to prenatal or intra-uterine causation, it is now well established that certain special causes, such as traumatisms, infectious diseases, and fright and other maternal impressions, may act through the mother. Several cases have been recorded in which autopsies have shown actual evidences of injury to the brain of the child during pregnancy. Recent clots or older cysts have occasionally been discovered. In one case, cited by Cotard, the mother had received a blow on the abdomen, and an old lesion was found in the brain of the child, which was stillborn and showed hemiplegic contractures. Gibbs has recorded another case of fetal cerebral hemorrhage apparently due to a blow received by the mother. All authorities are in accord as to the effects of a serious illness of the mother, such as infectious and septic diseases like typhoid fever, severe influenza, pneumonia, nephritis, and the exanthematous fevers. Osler records a case in which a large recent clot was found in the brain of a fetus six months old, the mother having died of typhoid fever. In some cases the child may suffer coincidently with the mother from infectious or toxic disease. Fright or any powerful emotional impression made upon the mother during pregnancy is a cause often alleged and occasionally effective, and the doctrine of maternal impressions receives some support from the records of rare cases of infantile cerebral paralysis. Hemor-

rhage into the brain, and softening from occlusion of vessels, while rare, are occasional fetal lesions. Fetal encephalitis and ependymitis have been suggested, but their occurrence has not as yet been fully demonstrated. Besides these more or less active prenatal causes, not a few of the cases are due to developmental arrests. The brain fails to develop because it lacks embryonal potentiality. Hereditary taint undoubtedly plays its part. In some instances several children in the same family are affected, although here certain mechanical conditions presented by the mother may have something to do with the production of the cases. While many of the cases seem to be clearly traceable to such causes as maternal impressions, injuries during pregnancy, birth before full term, difficult labor, and asphyxia, many children who are subjected to causes of this kind escape all forms of infantile rigidity and paralysis. The inherited weakness or predisposition being present, the exciting causes which have just been discussed must be given their proper weight. Inherited syphilis does not seem to play an important direct rôle in the development of these cases, although authorities differ upon this subject. Sachs traces hereditary syphilis in only one of the many cases which have fallen under his notice. It is not improbable that in some cases syphilis in the grandparents or in more remote ancestors may have some influence.

Etiology of Birth Palsies. Prolonged and difficult labor, particularly with firstborn children, is one of the most clearly demonstrated causes of cerebral palsies. While the direct application of force by instruments may inflict injuries upon the head and brain, these cerebral affections are far more frequently due to compression of the child's head in the pelvic strait, and to the asphyxia which results from protracted labor. Skilfully applied forceps, indeed, in some cases prevents the occurrence of these unfortunate palsies. As first clearly shown by McNutt, the actual lesion in most of the birth cases is meningeal hemorrhage, which is likely to be at the base of the brain in vertex presentations, and on the convexity in breech presentations. According to Peterson, spinal lesions, usually hemorrhage, in rare cases give rise to diplegias.

Etiology of Postnatal Cases. Among the causes most frequently operative after birth, the acute infectious diseases of infancy and childhood take first rank. Cases have been recorded as occurring during or after diphtheria, scarlet fever, measles, variola, varicella, whooping cough, mumps, typhoid fever, pneumonia, cerebrospinal fever, and influenza,—after almost every known febrile affection of childhood. In whooping cough hemorrhage may occasionally take place because of the violence of the paroxysms of coughing. Although the view of Strümpell as to focal polioencephalitis being a comparatively frequent cause of infantile cerebral palsies has received but little support, much can be said in its favor. Cases have

fallen under my notice in which the history of the attack preceding the occurrence of the paralysis certainly pointed to some acute cerebral inflammatory affection. In not a few cases close investigation shows a history of coma and convulsions, and fever is frequently present. Injuries to the skull and brain from falls and blows or wounds of various kinds are comparatively common causes. Some of the postnatal cases have been attributed to fright. As has been shown, convulsions are frequently present both in diplegias and in hemiplegias. Often they are concomitants, resulting from irritation or from the cerebral instability caused by the lesions which produce the paralysis and other phenomena of the disease. In comparatively rare instances the convulsions may give rise to hemiplegia or diplegia. When, for instance, they are due to severe peripheral irritation, like that of dentition, or to toxemia, as during infectious diseases, the influence of the convulsive attack may cause an actual destructive focal lesion of the brain, such as a hemorrhage, or it may inhibit the development of the brain. Sachs and Osler both attribute to convulsions this etiological influence.

Etiology of Special Clinical Types. Efforts have been made, and with some success, to relate particular clinical types to a special etiology. The hemiplegias as compared with the diplegias are certainly most frequently due to postnatal causes. Generalized rigidity has been shown in a large number of cases to be due to traumatisms during birth and to asphyxia; while paraplegic rigidity, which is in reality only a subtype of generalized rigidity, frequently follows birth before term. A number of these cases of rigidity have been found to be associated with meningeal hemorrhage situated along the median border of the cerebral hemispheres. The fact that meningeal hemorrhages are thus situated and affect both hemispheres, and that the centres for the lower extremities are near and on the mesal surface, accounts for the diplegias and for the frequent preponderance of rigidity and spastic paralysis in the lower extremities. While the arms are often affected, they are usually much less so than the legs. One arm is frequently much less affected than the other. These facts are to be explained by the degree of diffusion of the hemorrhage towards the Sylvian fossa, which diffusion is often greater on the one side than on the other. The face frequently escapes because of the lower position of its centres on the lateral aspect of the brain. The hemorrhage is usually due to rupture of small vessels, and especially of veins which empty into the longitudinal sinus, which accounts for the locality of the hemorrhage. When the effects of the hemorrhage are confined to the surface of the brain, rigidity largely predominates over paralysis. The depth of the cerebral injury in cases of traumatic origin chiefly determines the form of hemiplegia or diplegia. The deeper the destruction the more profound is the paralysis. When the hemorrhage is intracerebral and in the motor region, paralysis always

results. The choreic and athetoid cases, and particularly the former, are not often directly traceable to birth before term, difficult labor, or asphyxia. They are probably, in some cases at least, dependent upon fright to the mother during pregnancy. When the cases are of prenatal origin, the movements may be present with little or no real paralysis. In some cases the movements are imposed upon double hemiplegias or monoplegias, and are to be classed with those generally described as posthemiplegic disorders of movement. In a few cases a traumatism to the mother during pregnancy has apparently been the cause. Bilateral or double hemiplegia usually results from lesions occurring after birth. Cases with epilepsy are not often of intra-uterine origin, and when prenatal are probably, like the choreas, in some instances at least, dependent upon fright or other maternal influence. As has already been stated, epilepsies are more common in the hemiplegic than in the diplegic varieties of infantile cerebral palsies.

Pathological Anatomy.—Many of the facts regarding the general pathology and pathological anatomy of the affections under consideration have already been given when speaking of the varieties of diplegia and hemiplegia and of their etiology and pathogenesis, and also in the sections on hypertrophy, atrophy, and porencephaly, which includes the most important residual lesions present in the infantile cerebral palsies. The opportunity of determining the exact character and appearances of the intra-uterine initial lesions in the cerebral paralyses is seldom afforded. In rare instances, as in the cases recorded by Cotard, Gibbs, and Osler, gross fetal lesions, as clots or cysts, are discovered. In these prenatal cases congenital porencephaly (page 601) and forms of cerebral hypertrophy or atrophy, general or partial, are common (pages 594, 597). In some cases the fetal brain is unusually small and presents marked fissural and gyral peculiarities. Incomplete and defective development of the cells of the cortex may be everywhere present in some cases, constituting *cortical agenesis*, and, although all the layers and strata of the cortex may be largely lacking, the layer of great pyramidal cells is especially deficient when motor as well as mental arrest is prominent. It is not improbable, although not yet proved, that either infection or traumatism may give rise to focal or diffuse prenatal encephalitis. No facts have been recorded indicating the actual appearances of the initial lesions in such cases. They would probably be softened and hemorrhagic areas with ruptured vessels, leucocytes, granular cells, and neurogliar proliferations. In one case of cerebral diplegia and bilateral athetosis, recorded by Putnam, the pathological conditions found on autopsy were two purulent cavities, caseous degeneration, softening, and sclerosis. Such residual lesions point to previous diffuse inflammation as at least a portion of the initial morbid process. In the birth palsies which terminate fatally, clots

are usually found covering a large part of either the convexity or the base of the brain. Several years since, I made an autopsy in a case of stillbirth, three successive children of the same mother having died during or immediately after delivery. In this case a large meningeal hemorrhage was found occupying the longitudinal fissure and covering most of the convexity of both hemispheres. In one of McNutt's cases the autopsy showed a large clot covering the convexity of the posterior half of the left hemicerebrum, and in another of her cases the right hemicerebrum was found covered by a clot which spread into the central fissure and had destroyed the cerebral tissues as far as the ependyma of the lateral ventricle. Kundrat has described the occurrence of minute venous hemorrhages which he refers to compression of the longitudinal sinus, but Sachs doubts whether such hemorrhages can be connected with the production of infantile cerebral palsies. It has been suggested that compression of the head in prolonged labor causes hyaline degeneration in the vessels. On microscopical examination, fatty degeneration of the walls of the encephalic vessels, a condition first described by Recklinghausen, is sometimes found. Such diseases of the bloodvessels should always be looked for microscopically. As stated on page 445, Meigs found typical chronic endarteritis in a marasmic child who died at the age of five months. In a specimen of brain cortex removed for Sachs by Dr. Gerster—the case having been one of hemiplegia with epilepsy in a child twelve years old—microscopical examination showed thickening of the pia, an increased number of thickened arteries, and large pyramidal cells, misshapen, granular, and diminished in number. Out of seventy-eight cases of infantile hemiplegia with autopsies, studied and tabulated by Sachs and Peterson, twenty-three were attributed to hemorrhage, seven to embolism, and five to thrombosis. These included cases occurring at and after birth. Osler has summarized some of the more important facts regarding hemorrhage, embolism, and thrombosis as initial lesions. In five cases heart disease was associated with embolism of the right medicerebral artery; in four cases hemorrhage was present; the right Sylvian artery was plugged by a firm thrombus in one case; in another a hemorrhage into the longitudinal fissure had been caused by a rupture of an aneurism of the right precerebral artery; and ventricular hemorrhage was found in still another case. The appearances presented by acute softening from embolism or thrombosis are similar to those which have already been described in discussing arterial embolism and thrombosis in adults, qualified somewhat by the different texture of the fetal and infantile brain. Disseminated foci of tuberculosis have been found in some comparatively recent cases. With regard to the appearances presented by cases with focal or diffuse encephalitis, the remarks on the pathological anatomy of these affections should be consulted (page 534 and pages 537, 538).

Diagnosis.—*Differentiation of Cerebral and Spinal Cases.* The diagnosis of cerebral infantile palsies must in the first place be made from those which are of spinal origin. Most important helps are afforded by the history of the onset, the distribution of the paralysis, the mental condition of the patient, the presence or absence of tonic and clonic spastic phenomena, and a study of the electrical reactions. The cerebral cases frequently originate before birth or during labor, while spinal cases are rarely congenital and still more rarely occur at the time of birth. In the cerebral birth cases, and in those which are postnatal, usually the history of the onset is one of asphyxia, convulsions, and coma, with pronounced hemiplegia or diplegia if paralysis can be determined. Hemiplegias occurring during the early years of life have the history of some form of apoplectic attack, which may be that of hemorrhage, embolism, thrombosis, abscess, tumor, or even localized tubercular meningitis, the symptoms of which affections have already been considered. In poliomyelitis of spinal origin the history is more likely to be that of an acute febrile attack of greater or less severity ; but it is true, as noted by Strümpell, that spinal and cerebral cases are sometimes ushered in by similar symptoms, which is perhaps to be explained by the fact that they are often due to infections or toxemias which act on the nervous system, exerting their destructive influences only on localized areas in the interior of the brain and the spinal cord. In the cerebral cases the paralysis is of one of the diplegic or hemiplegic types ; while in spinal cases it is more frequently limited to one or two limbs, or to groups of muscles in one limb, at least so far as the persistent symptoms are concerned. In cerebral cases spastic phenomena largely predominate, in spinal poliomyelitic cases the paralysis is flaccid. In deciding between the spinal and the cerebral cases stress should be laid on the mental condition of the patient, which is absolutely or comparatively good in the former. Choreic and athetoid phenomena point to a cerebral disease. Convulsions are occasionally present at the onset of a case of infantile spinal paralysis, but they are not likely to recur. Convulsions are of frequent occurrence at the onset and during the course of cerebral cases. The tendon and muscle phenomena are exaggerated in cerebral cases, and, as a rule, diminished or lost in spinal cases. In the spinal poliomyelitic cases the electrical reactions are those of degeneration. The presence of such reactions indicates spinal or neural disease. Vasomotor disturbance, muscular atrophy, and other trophic changes are more marked in paralysis due to anterior poliomyelitis ; but groups of muscles, a limb, or the entire half of the body may be much diminished in size in cerebral cases. Erb and others at one time held that most of the spastic paraplegias were of spinal origin, and the fact that in rare cases they may be of this origin is generally recognized.

Differentiation of the Family Forms of Spastic Paralyses. In the

spinal family cases spastic paralyses are probably, as a rule, dependent upon arrest of development of the spinal portions of the pyramidal tract. Of the family forms of spastic paraplegia, to be hereafter considered, some are cerebral, some spinal, and some cerebrospinal. In two cases, brother and sister, which came under my observation, the girl presented symptoms of spastic paraplegia from birth, while her brother began to show evidences of the affection only after the age of puberty, and did not lose the use of his lower limbs entirely until he was almost thirty years old. In both of these cases the intelligence was unimpaired. Occasionally a sporadic case of spinal spastic paraplegia is observed, as one recorded by Sachs. This child, who was four years old when first examined, presented paresis and rigidity of both lower extremities, increased knee jerk on both sides, slight double ankle clonus, normal electrical reaction, no disturbances of sensation, and fair intelligence. Strümpell has described one form of hereditary spastic paralysis, the symptoms of which do not usually show themselves until the third decade of life; but these, of course, need not be considered in connection with the infantile cerebral diplegias, except to note the fact of their occurrence, and to remark that they may be related to the forms which occur congenitally or earlier, the pathology being similar.

Association of Cerebral and Spinal Lesions in the Same Case. When the case is apparently of cerebral origin and the conditions which belong to spinal cases are present, careful inquiry should be made in order to discover whether the child has not suffered from a spinal instead of or in addition to a cerebral affection. In one case recorded by Sachs the reflexes were increased in the upper extremities and decreased in the lower, although the case was one of right hemiplegia. On close examination it was discovered that two years after the onset of the cerebral palsy the child had suffered from an attack of poliomyelitis. A cerebral and a spinal paralysis may be present in the same case; or both forms of paralysis may originate or appear to originate from the same causes. In one of my own cases a typical spinal paralysis was present although the history and symptoms seemed to point to a cerebral lesion. The birth of this child was instrumental, and two hours after birth it was seized with convulsions which persisted for twelve hours. At the points where the forceps had been applied, evidences of injury were present in the frontal bones in the form of marked depressions, which have remained. When the child was nine months old it was for the first time noted that the left leg was smaller and was not properly used. The child had a second attack of convulsions when he was sixteen months old, and another at the age of three years. He was five years old when brought to my Polyclinic service. In the left leg all the muscles supplied by the internal popliteal nerve were paralyzed or their movements much impaired. The limb below the knee was atrophied, with loss of

faradocontractility in the affected muscles. The tendo Achillis was a thin narrow ribbon, and the foot assumed the talipes calcaneus position. The skin over the paralyzed muscles was scurfy and cold. Knee jerk was absent. No sensory changes were present.

Differentiation of Pseudoparalytic Rigidity and Spastic Diplegia. The diplegias need sometimes to be differentiated from forms of pseudoparalytic rigidity. This is an affection which is often associated with rickets or other constitutional disturbances. Osler has tabulated the chief differences, as follows:

Pseudoparalytic Rigidity.	Spastic Diplegia and Paraplegia.
Follows a prolonged illness. Is often associated with rickets, laryngismus stridulus, and the so-called hydrocephaloid state.	Usually exist from birth. History of difficult labor, of asphyxia neonatorum, or of convulsions.
Begins in hands as carpopedal spasm; often confined to hands and arms.	Arms rarely involved without legs, and not in such marked degree.
Spasms painful, and attempts at extension cause pain.	Usually painless.
Intermittent and of transient duration.	Variable in intensity, but continuous.

Rachitic Pseudoparaplegia and Tetany. Under the designation rachitic pseudoparaplegia Berg has described an affection in which the patients are often unable to walk or are extremely weak, and it may be supposed that these cases of diplegia or paraplegia are due to destructive central lesions. The diagnosis will be assisted by the discovery of the fact that these pseudoparaplegics have the signs of rachitis, as shown in the sternum, spine, liver, spleen, and other tissues and organs. Close examination shows that the muscles are not really paralyzed or atrophied. The electrical reactions are normal, and these patients usually improve and become able to walk under the influence of fresh air, good food, and the administration of cod liver oil, and preparations containing iodine, such as Lugol's solution, syrup of iodide of iron, and hydriodic acid. While rachitic cases of this type may improve or recover, it must, however, be remembered that some true diplegics are also rachitic, so that the presence of rachitis is not a proof that the patient is not also a sufferer from an organic cerebral lesion. All the facts in such a case must be carefully weighed before advancing a positive opinion. Tetany, which is sometimes associated with rachitis, for a brief time may suggest one form of spastic diplegia, but such an error is not likely to be made and would probably soon be corrected. A consideration of the previous history of the patient, the paroxysmal character of the affection, the suddenness of its onset, the peculiarities of the spasm, and the etiological points serve to make the diagnosis of tetany clear.

Infectious and Postinfectious Paralyses. As infectious paralyses may be either of cerebral, spinal, or peripheral origin, or may

represent various combinations of cerebral, spinal, or peripheral disease, it may be at times difficult to differentiate the types of organic diplegia and hemiplegia under consideration from other forms of infectious or postinfectious paralysis. These infectious paralyses have been thoroughly considered in a paper by Bassette, who has shown that they occur in connection with measles, diphtheria, scarlet fever, whooping cough, mumps, influenza, malaria, and typhoid fever, and include cases of myelitis, multiple neuritis, diffuse or limited neuritis, and neuritis combined with myelitis, besides a few cases of distinctly cerebral type commonly due to embolism, hemorrhage, or thrombosis. As is well known, some of these postinfectious cases may show widespread paralysis, involving both upper and lower extremities and the ocular muscles, and such cases might without due consideration be regarded as more intractable types of diplegia. In most of them the age of the patient, the history of the acute infectious attack, and the evidences of peripheral or spinal involvement as shown by the electrical reactions, the tendon and muscle phenomena, and other signs of neuromuscular inflammation or degeneration, are sufficient to make the diagnosis.

Tumors. A case of infantile cerebral paralysis may of course be due to tumor, a localized meningitis, an abscess, or other active lesion more or less amenable to treatment, and it is therefore important to separate these cases from the forms of diplegia and hemiplegia now under consideration, which are commonly due to other causes. The rules of diagnosis given in the previous sections for tumors, abscess, etc., will answer here, but it is desirable to remember a few special points. Tumors of the quadrigeminal body, of the pons, or of the cerebellum sometimes give rise to diplegic rigidity of more or less regular form, the cerebellar cases in which these manifestations are present being usually so situated that they compress or irritate the pons or postoblongata. Bilateral neoplasms symmetrical or nearly symmetrical in situation, and partly cortical and partly subcortical, might give rise both to diplegic spasticity and to convulsions of confusing type. Single tumors situated in the motor cortex or subcortex may in children as in adults be the cause of spastic hemiplegia and Jacksonian epilepsy.

Peripheral Obstetrical Paralysis. Certain forms of peripheral paralysis of the face or extremities which were termed by Duchenne "obstetrical paralyses" may originate from the direct application of force by traction by the forceps. Either the face or one of the arms is usually affected. Investigation of such cases will soon reveal that they are peripheral; and the diagnosis will be assisted by the absence of such symptoms as coma, convulsions, rigidity, and spastic paralysis involving more than one part of the body. The paralysis is usually of a flaccid character and limited to one member or to one side of the face.

Differentiation of Prenatal, Natal, and Postnatal Cases. As regards the differential diagnosis of congenital or fetal cases of diplegia or hemiplegia from those originating during labor or after birth, most of the points of value in their separation have already been given when discussing the different clinical and etiological varieties. A case apparently postnatal may have begun during intra-uterine life, a point to which attention was called when speaking of congenital and acquired porencephaly. Pronounced idiocy is in favor of an intra-uterine etiology, although lower grades of idiocy, imbecility, and feeble-mindedness may be present both in the birth palsies and in those which originate during the first years of life. Asymmetries and special deformities of the cranium, unless they are such as have evidently been produced by instruments during delivery, point to an intra-uterine rather than to a birth paralysis. The shape of the head, like that of the brain, is in these cases determined by congenital causes. A study of facial expression may be an important aid to diagnosis in young infants, in whom it is often difficult to determine the existence of diplegia and imbecility by an investigation for paralysis and rigidity or by general efforts at gauging the child's intelligence. A peculiarly morbid and suggestive appearance is sometimes given to the face, as when the mouth is kept open by the overaction of the radiating on the circular muscles. The child may have the appearance of weeping when it would laugh, or the reverse. The fact that a child is born asphyxiated does not prove that the case is of obstetrical origin. A child may also be born prematurely because it is defective, and the case be supposed to be one due to birth before term. While convulsions are frequently present in natal and postnatal hemiplegias, considering only the diplegias, the presence of epilepsy is, on the whole, in favor of congenital origin. Between diplegias occurring at the time of birth and those which originate later the difficulties of differentiation are sometimes great. Generalized rigidity and paraplegic rigidity are most frequently due to birth lesions. The fact that a child has apparently been born healthy, and that the paralytic or diplegic affection has come on at a definite time and in association with a special attack, is of course of value in separating the cases occurring after birth from natal and prenatal cases. Certain signs or symptoms may remain latent for days, weeks, or months after birth. Epilepsy may thus remain latent, and even the paralysis may not show itself positively for some time.

Adenoid Vegetations in Infantile Hemiplegics and Diplegics. It has several times come to my notice that the presence of adenoid vegetations in cases of idiocy and imbecility, with or without paralysis, has led to an error of diagnosis or of prognosis. These vegetations are somewhat commonly present in such cases, and the throat specialist is led to believe that by their removal he can remedy the defects of

speech and get rid of what he regards simply as apathy or stupidity due to local lesions. The habit which many diplegics have of holding open the mouth favors the occurrence of such vegetations, which should be properly treated. Some of the special disorders of respiration and digestion from which such patients suffer may be relieved by proper treatment of the adenoids, and choreic and athetoid movements have also been reported as thus relieved.

Prognosis.—It is best to separate the consideration of the prognosis of the diplegias from that of the hemiplegias, and particularly from the hemiplegias of postnatal origin. The prognosis of by far the largest number of diplegias is unqualifiedly bad. The paralytic, spastic, and other physical conditions are grave, and the patients usually present a greater or less degree of imbecility or idiocy. In cases of infantile cerebral paralysis acquired after birth it may be difficult to give a prognosis as to the amount of improvement or recovery that will take place until one or two years have elapsed. A very few cases make almost complete recovery; others, also few in number, remain partially paralyzed, but with fair or even good mental power; a large number develop epilepsy and various grades of mental retardation. Convulsions may be present at first, may disappear for weeks, and may return after months or even years. In other cases they may appear for the first time several months or even years after the onset of a hemiplegia. The presence of convulsions always adds to the gravity of the prognosis. Cases with choreoid and athetoid movements are also of especially unfavorable prognosis. In rare cases patients are met with who present only moderate or even slight degrees of generalized or paraplegic rigidity and are so nearly normal that they are scarcely recognized as diplegics. Cases of paraplegic rigidity or of spastic paraplegia are relatively of more favorable prognosis than those in which the upper as well as the lower extremities are involved. Mentally as well as physically these cases are usually less ominous. The double spastic hemiplegias sometimes make slight improvement, and in some instances even considerable improvement, over the conditions first observed in infancy. The bilateral choreas either remain stationary or get progressively but very slowly worse. It may be difficult or impossible to answer with positiveness the question put to the physician as to whether a diplegic child from two to five years old will be able to walk. Such children do sometimes become able to stand or even to walk after they are five or six years old, although their gait is spastic or may be of the cross-legged type. In postnatal cases the younger the child the more probable is it that serious mental deterioration will be left. In the hemiplegics the leg and the face nearly always improve much more than the arm, so that frequently the patient will regain the power of walking and may be left with little or no asymmetry of the face. Most cases of diplegia die in childhood or before

the age of adolescence. The hemiplegics have a longer average lease on life, and occasionally reach middle age. In all cases of convulsions in children care should be taken to note the subsequent condition of the child, and in some instances parents should be warned that a paralytic or mental disorder may be left.

Treatment.—*Treatment of the Initial Period and of Special Symptoms.* In prolonged and difficult labor the skilful use of the forceps may prevent the occurrence of a disastrous meningeal hemorrhage; but if such hemorrhage should occur and be recognized as present, the question of immediate surgical interference may arise. Opening the skull to remove the clot in a case of meningeal hemorrhage at the time of birth has not, so far as I know, been practised, but, as such a case usually either terminates fatally or leaves serious permanent paralysis, the procedure would in some cases be justifiable. Great care, of course, should be taken to avoid injuring the delicate brain of the child. When an infant has had a serious fall or has received a blow on the head and a hemiplegia or monoplegia follows, at the present day the tendency is to have recourse promptly to trephining. This in some instances is the correct thing to do, especially as antisepsis has rendered such operations comparatively safe, but oftener it is better to wait. Osler refers to two cases of speedy recovery without operation, one in his own practice and one in that of Murray Cheston. In the former a child twenty-three months old had fallen from a balcony and had a large hematoma of the scalp, with left hemiplegia and coma. The symptoms gradually disappeared, and complete recovery followed. Murray Cheston's case was that of a child who was tripped up by his brother and fell on his head. There was no external wound or fracture, but hemiplegia developed, which completely disappeared in a few days, leaving the boy entirely well. Such cases may be due to bruising of the brain with little or no hemorrhage. In a case of traumatism, if, in addition to the paralysis, convulsions and other signs of irritation are present and persist, trephining for removal of the clot should be resorted to after three or four days; but if spasm is absent or soon ceases, and if the paralysis begins to disappear after two or three days, operation should be postponed. When convulsions are present at or soon after birth the child should be kept absolutely quiet, cold applied to the head and heat to the extremities, and small doses of calomel administered. Either the bromides or chloral in small doses should be given, and repeated if necessary. One grain of chloral by the rectum, or a less amount by the mouth, will be sufficient; and any of the bromides can be used in doses of from one to three or four grains. The careful inhalation of chloroform may be resorted to to check the convulsions when one follows another in a threatening series. A few drops of the anesthetic can be sprinkled on a soft cloth and cautiously administered. If the convulsions cease, careful watch should be kept,

and the inhalations repeated at the first threatenings of a return. After the immediate effects of the attack have passed off, small doses of iodide and bromide should be given for several weeks, careful attention being paid to the digestive organs of the child. In infants the amount of iodide should not exceed from one half to two grains. A good method of administration is to give sodium iodide in milk. The bromides can also be given in doses of from two to eight grains. Minute doses of Fowler's solution of arsenic may be combined with them. The bromides are particularly called for when the convulsions show a tendency to recur. When hereditary syphilis is suspected, small doses of mercurials or of the iodides may be carefully used. If the child shows evidences of rachitis, cod liver oil should be given, either internally or externally, and with it, or alone, preparations containing iodine, such as Lugol's solution, syrup of iodide of iron, and syrup of hydriodic acid, may be employed,—of course in minute doses. The emulsions of cod liver oil containing lime are particularly efficient. Antirachitic treatment is especially desirable in some cases because, as shown under diagnosis, a rachitic pseudoparaplegia or a pseudoparaplegic rigidity associated with rickets sometimes may be mistaken for a true diplegia. The treatment in such cases, therefore, may assist in clearing up the diagnosis, and can do no harm, even if it does not do good. The greatest care should be exercised as to warmth and exposure with infants born prematurely.

Treatment of the Residual Conditions and Symptoms.—Faradism or galvanism, massage, Swedish movements, or systematized exercises, may do good in a few instances. Electric mechanical and other peripheral stimuli may serve to call out some latent power, but care should be taken not to overtreat. The general health should always be maintained at as high a point as possible. For the athetosis or athetoid movements often present little can be done. Nerve stretching may stop the movements for a time, but the relief is seldom sufficient to warrant the operation. In one case of severe athetosis referred to by Peterson the arm was removed, with great relief to the patient. The persistent athetoid and choreic movements have in some instances been temporarily relieved by mechanical and surgical measures, and especially by the application of splints and plaster bandages. Congenital diplegics will usually not have much capacity for improvement by mental training. Relatively the cases which occur at birth and the postnatal cases, especially the latter, yield better results. A child with a normal brain may be the victim of a meningeal hemorrhage during labor or at any period after birth as the result of a traumatism. The amount of damage done by such active lesions will largely determine the degree of improvement that can be accomplished by subsequent training. Possibilities may in some of these cases be neglected if training is not attempted. In the cases

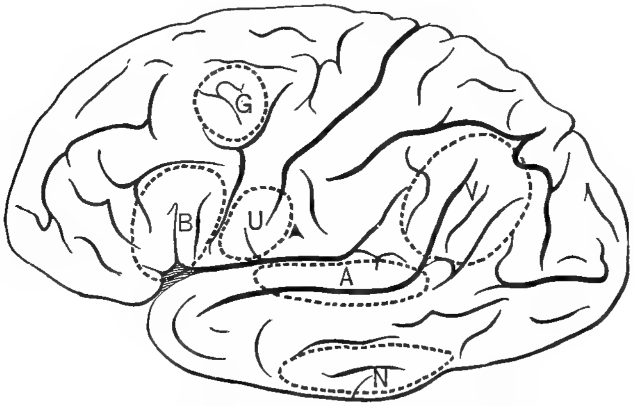
acquired one or more years after birth, a process of reeducation of the brain may be needed. For defective speech as well as for general mental deficiency systematic and persistent efforts at training should be tried.

Surgical Measures. Efforts have been made, chiefly surgical, to treat the porencephaly, but from the very nature of the lesion such treatment is usually futile, and may be harmful. An absence of cerebral substance—a hole in the brain—is not to be cured or even relieved by making an opening into that hole and draining its contents. If the fluid is removed from the cavity it will fill up again; and if efforts are made to prevent this by drainage, death in all probability will soon result. In addition, the patient will be subjected to the danger of infection from without. The emptying of brain cysts is in all cases a doubtful procedure, and it is more than doubtful in porencephaly. Operations in such cases are not only most unsuccessful, but are often fatal. If the walls of an old hemorrhagic or other cyst are a source of irritation, it is conceivable that their removal may diminish this irritation and lead to improvement. Even in old cases of fracture, tumor, and localized meningitis or hemorrhage, trephining does not offer much hope, but operation here has a more reasonable basis and may sometimes be of benefit. In carefully selected cases something can be done to improve the station and gait of diplegics and hemiplegics, most frequently the former, by operations for the relief of the contractures. In cases of extreme rigidity and paralysis with imbecility or idiocy nothing can be accomplished by such procedures, which are sometimes resorted to without due consideration. The child's mental condition is such as to prevent advantage being taken of the results of operation; and the corticospinal and peripheral motor apparatus is so arrested or degenerated that the limbs tend to return to their former positions. When the patient has sufficient intelligence to submit to advice and training, and when he already has some control over the movements of his limbs, some benefit can be obtained by cutting carefully selected tendons. The tenotomies may enable him to get his feet to the floor better, or to assume less constrained positions in standing or walking, or to make somewhat better use of certain groups of muscles over which he has some voluntary control, but too much must not be expected. Photographs of such patients before and after operation, which seem to show that great improvement has been attained, are often simply striking as pictures of still life. I would not altogether discourage operations in such cases. In extreme contracture much constitutional disturbance may be produced as the effect of operation, and death may result. The most generally useful of tenotomies is the simplest, namely, that of cutting the tendo Achillis in order to get a plantigrade walk. Neurectomy has been performed with reported success in some cases of contracture.

APHASIA AND OTHER DEFECTS OF SPEECH DUE TO DISEASE OF THE CEREBRUM.

Cerebral Centres and Tracts concerned in Speech.—The cortical centres concerned in speech are (1) the *primary* and *secondary cortical visual centres;* (2) the *primary* and *secondary cortical auditory centres;* (3) the *naming* or *concept centre;* (4) the *motor speech centre;* and (5) the *motor graphic centre.* The cortical centres are united with each other by associating tracts, those on the sensory side are connected with the basal receptive centres, and those on the motor side with the basal emissive centres. Higher inhibitory centres are situated in the prefrontal region. For the discussion of the situation and special functions of these centres the reader is referred to the section on cerebral localization in Chapter V. In Fig. 331 are shown the locations of the various centres enumerated in accordance with the views of the writer. A diagram of the speech centres and tracts is given in Fig. 332. These are, in the first place, the tracts which pass from the basal auditory and visual centres to the primary cortical centres for hearing and vision. In the second place, they are the tracts which pass from the primary cortical centres for hearing and vision to the secondary or higher cortical centres for word hearing and word vision,—it being probable that these tracts converge from the primary cortical centres of both hemispheres to the secondary or higher cortical centres of the left hemisphere. In the third place, they are the tracts which pass from the higher auditory and visual centres to the naming or concept centre: in the diagram only the tract from the auditory centre to the naming centre is represented. Other tracts are that between the concept centre and Broca's centre

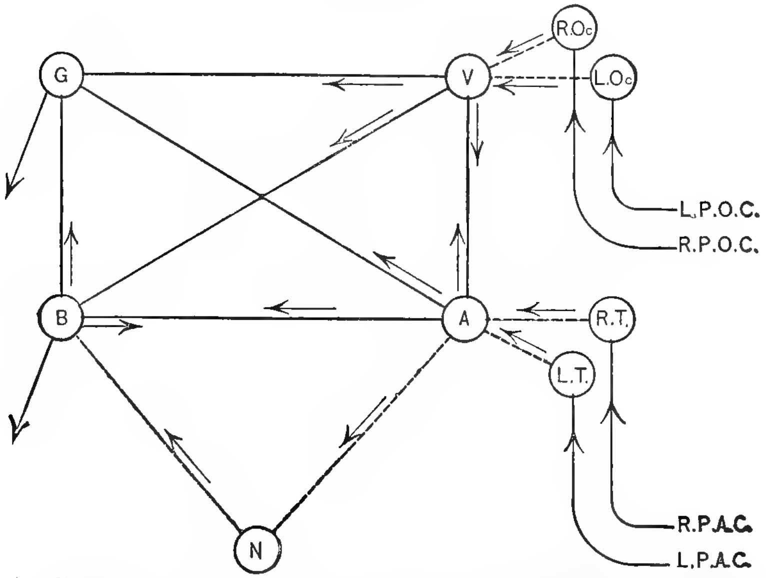
FIG. 331.



A, auditory centre (centre for word hearing); V, visual centre (centre for word seeing); N, naming centre (centre where percepts are given a name); B, motor speech centre (in Broca's convolution); G, graphic centre; U, utterance centre.

in the left third frontal convolution; the direct tract, sometimes used, between the auditory centre and Broca's centre; the direct tract, sometimes used, between the higher visual centres and Broca's centre; the direct tract, sometimes used, between the higher visual centre and the motor graphic centre; and the tract or tracts connecting the motor cortical centres concerned in speech and writing with the centres in the bulb and spinal cord. Tracts also associate the visual and auditory centres, and the motor speech and motor writing centres. These tracts are shown in the diagram and are

FIG. 332.



A, auditory centre (centre for word hearing); V, visual centre (centre for word seeing); N, naming centre (centre where percepts are given names); B, motor speech centre in Broca's convolution; (an utterance centre, U in Fig. 331, is also required to complete the motor side of the speech process;) G, graphic centre; R. Oc., primary cortical visual centre in the right occipital lobe; L. Oc., primary cortical visual centre in the left occipital lobe; R. P. O. C., optic centres at the base of the brain, right side; L. P. O. C., optic centres at the base of the brain, left side; R. T., primary cortical auditory centre in the right temporal lobe; L. T., primary cortical auditory centre in the left temporal lobe; R. P. A. C., auditory centres at the base of the brain, right side; L. P. A. C., auditory centres at the base of the brain, left side.

explained in the legend, the direction in which impulses are conveyed being indicated by the arrows. Much more than a mere knowledge of the positions and functions of these cortical centres is necessary in order to understand the cerebral mechanism of speech. The student should have a thorough knowledge of the types and subtypes of aphasia, and of the terms descriptive of them. He should know how to diagnosticate one cortical disorder from another, and cortical from subcortical affections. He should be able to forecast partial or complete recovery, should comprehend the bearing and significance of pantomime, and should be familiar with the

methods of studying aphasic subjects and with the best methods of medical, surgical, and educational treatment. The important relations of cerebral disturbances of speech to mental phenomena require his attention both for medical and medico-legal reasons.

Definitions and Synonyms.—Aphasia is the loss or impairment of the power of comprehending or expressing ideas by language or signs ; and, as now commonly employed, the word is a general designation applied to any or all of the defects of speech dependent upon disease of the cerebral hemispheres. The word aphasia was introduced by Trousseau, and, strictly construed, means loss of speech. No synonyms have succeeded in displacing it. McLane Hamilton suggested *asemasia*, which means the inability to communicate by signs or language, and etymologically this is an excellent term. *Aphemia* was used by Broca and others, and is still sometimes employed in the description of cerebral speech disturbances in general, but is used mostly to describe the commonest forms of motor aphasia. Bastian has suggested that aphemia be restricted in its definition to defects of speech from lesions of those fibres which connect the so-called motor centres of the cerebrum with the bulbar nuclei,—that is, to subcortical motor aphasias. *Alalia*, employed by Lordat in the general sense afterwards accorded to aphasia, is now commonly restricted to the meaning given to it by Kussmaul, who defines it as an entire inability to utter articulate sounds, placing it under defects of enunciation. The proper usage of *amnesia* is to express loss of memory. With certain qualifying adjectives it correctly describes special varieties of aphasia, as *verbal amnesia* and *literal amnesia*. *Amnesic aphasia* as a general term has been applied to disorders of speech from lesions on the receptive or sensory side of the brain; and *ataxic aphasia* is another old term, still often used to describe the motor aphasia of common type.

Anatomicopathological Varieties of Aphasia.—From the anatomical standpoint aphasias can be subdivided into cortical, subcortical, and transcortical. A *cortical aphasia* (pictorial aphasia of Wyllie) is one due to a lesion of the cortex; a *subcortical aphasia* is one dependent upon a lesion of the subcortical projection fibres, sensory or motor; and a *transcortical aphasia* is the result of disruption of the tracts which associate together the various sensory, motor, and conceptual cortical centres. Two varieties of subcortical aphasia are recognized by Wernicke, Wyllie, and others. The first is *subcortical sensory aphasia*, in which the affection is due to a lesion of the converging incoming tracts to the higher sensory centres. These tracts are those which lead to the auditory, visual, tactile, and other cortical receptive centres, but, as a rule, only the incoming auditory and visual paths are considered in discussing the subject. *Subcortical auditory aphasia* is a variety of subcortical sensory aphasia, and is due to a lesion of the tract which passes

from the centres for general audition to the higher cortical auditory centre in the left hemisphere,—the centre for word hearing. *Subcortical visual aphasia* is due to a lesion of the tract which passes from the centres for general vision, or primary cortical visual centres, to the secondary or higher cortical centre of the left hemicerebrum,—the centre for word vision. The second form of subcortical aphasia is *subcortical motor aphasia*, a disorder which is due to a lesion of the tracts connecting the cortical centres for motor speech with the bulbar nuclei. Subcortical sensory and motor aphasias are sometimes spoken of as *infrapictorial sensory aphasias* and *infrapictorial motor aphasias*, because they are the results of lesions of parts of the brain below or beneath the centres where the sensory and motor images of words are revived. A subcortical motor agraphia is possible,—an agraphia due to lesion of the tract which passes from the graphic motor centres to the nuclei in the cervical cord which are related to the neuromuscular apparatus concerned in writing. Transcortical aphasias are of as many kinds as there are associating tracts. They are sometimes spoken of as *suprapictorial sensory aphasias* and *suprapictorial motor aphasias.* The transcortical sensory aphasias are those resulting from severance of the paths between the sensory and concept areas; the transcortical motor aphasias are those resulting from the cutting across of the path or tract between the concept and motor speech areas. Transcortical sensory aphasias may be either of an auditory or a visual type, the former being due to lesion of the tract between the auditory and concept centres, and the latter to lesion between the visual and concept centres. Transcortical sensory agraphia is an affection due to lesion between the visual and graphic centres; while a transcortical motor agraphia is one dependent upon a lesion of the tract between the concept and graphic centres. In some cases sensory centres and associating fibres, or motor centres and associating fibres, or both sensory and motor centres and associating fibres, may be involved in the same lesion, giving rise to different forms of *combined* or *mixed* aphasias. An extensive lesion may cause a *total sensorimotor* aphasia. The term *pure* is sometimes applied to some of the varieties of aphasia above described, as to subcortical auditory aphasia, subcortical visual aphasia, and subcortical motor aphasia. The affection of speech in these cases is due to a lesion of the tracts leading to the cerebral percept centres or passing from the cerebral motor centres concerned in speech. The mechanism of interior language is not impaired in the pure aphasias, which are due to a blockage or an interruption of the way in or the way out.

Clinical Varieties of Aphasia.—Three main clinical varieties of cortical and subcortical aphasia may be recognized,—*sensory aphasia*, *conceptual aphasia*, and *motor aphasia*. By some authors both sensory and conceptual aphasia are included under sensory aphasia. We

may have various forms of sensory or motor aphasia, according to the location of the lesions. Among the forms of sensory aphasia, for instance, are verbal blindness and verbal deafness and their subtypes, and psychic blindness. Concept and motor aphasias are also of several forms, and we may have mixed sensory and concept or mixed concept and motor aphasias, according to the parts implicated in the lesions. These different clinical varieties of aphasia will now be separately considered.

Verbal Deafness.—*Definition, Varieties, and Symptoms.* Word deafness, or verbal deafness, is the inability to understand spoken words. In word deaf patients the external and internal apparatus for hearing may be intact, as may also be the centres and tracts concerned with hearing below the cerebrum. The term auditory aphasia is preferred by some authors, as by Wyllie, who thinks that verbal deafness is too narrow a designation, but he evidently includes under his auditory aphasia some affections which should be considered separately from word deafness. Verbal deafness may be complete or incomplete. It is not infrequently complete at first and gradually recovered from in large part. Sometimes several categories of verbal deafness are made, as (1) that form of deafness in which the patient recognizes the voice as a noise, but as nothing more; (2) that form in which he recognizes language as such, but does not understand what is said; and (3) that form in which language is heard and can be repeated, but in which the patient does not understand what he repeats. (Brissaud.) These different varieties may merge into one another, giving irregular types of word deafness, which call for the most critical study. A patient suffering from word deafness does not understand spoken language, unless in occasional instances he is able to recognize his own surname, more rarely his given name, and perhaps a few other words. If he is not otherwise deaf, he recognizes ordinary sounds. He is like one who hears a language of the meaning of which he has not the slightest idea. (Dejerine.) He is also unable to read aloud correctly, and, if the verbal deafness is complete, he cannot echo the words of others. If the case is one unassociated with verbal blindness, the patient may be able to read, and perhaps he can even read aloud, but his reading will show paralexia, words and syllables being confused and jumbled in their articulation. He is not able to verify what he reads by his sense of hearing. The position of the centre for word hearing, lesion of which causes the cortical variety of verbal deafness, has already been sufficiently considered (see pages 344 and 345 and Fig. 232). The centre is situated in the caudal portions of the first and second temporal convolutions, the chief part in the process probably being played by the first temporal convolution.

Pure Word Deafness. A few cases of total verbal deafness not due to lesion of the auditory centre have been recorded. These are illus-

trations of the subcortical auditory aphasia which has already been defined. Lichtheim, for instance, has recorded a case of pure word deafness in which the patient preserved the power of volitional speaking, of writing, and of reading aloud. He had neither paraphasia nor paragraphia. Sérieux has reported a case similar to that of Lichtheim. Such a case is probably due to a lesion of the tract or tracts from the primary cortical auditory centre or centres to the secondary cortical auditory centre (the centre for word hearing). A lesion of this tract causes word deafness, as does also one of the centre for word hearing, but in the latter case paraphasia and paralexia are present, because the patient is unable to verify through his centre for word hearing what he says or means, whereas in the former case he can do this. In this form of word deafness the patient does not understand the questions asked him, and is not able to write from dictation. These are the only symptoms which he presents. He understands writing,—which is the only means of entering into communication with him,—and replies correctly in a loud voice to the questions which are asked him on paper. Spontaneous writing and copying are normal, and intelligence is intact. The patient is not the subject of verbal amnesia nor of other forms of speech disturbance. The intact centre for word images is cut off from the lower centres and the outside world. The patient may hear the words as sounds although not understanding what is said.

Mixed Sensory and Concept Aphasia.—In some cases of word deafness verbal amnesia is also present, but this may be due to involvement of the tracts leading from the centres of hearing to the centres for concepts or to the motor centres, or in some instances to lesion of the naming or concept centre itself. A lesion of the tract leading from the centre for word hearing to the concept centre, as already stated, gives what is anatomically termed transcortical or suprapictorial auditory aphasia. In this transcortical auditory aphasia, incoming audible speech is not understood, nor, according to Lichtheim and Wernicke, is incoming visual speech, as the path between the auditory centre and the naming or concept centre is cut across, so that the auditory word pictures revived in the auditory centre do not call up the ideas or meanings in this higher centre. What is heard or read is not understood, and yet the word images in the auditory centre are intact and can be revived from without. Whatever is heard can be repeated, and the patient can easily read aloud, but he understands neither what he hears nor what he reads. He can write to dictation, and he can copy from print or from writing. Echo speech is a characteristic symptom. Amnesia for nouns, paraphasia, and, in consequence of the latter, paragraphia, are also shown. Lichtheim has suggested the name inner commissural word deafness for this variety.

Verbal Blindness.—*Varieties.* Verbal blindness is the inability to understand by sight written or printed words. It may be subdivided into *verbal blindness proper* and *literal blindness* or blindness for letters. Verbal blindness is occasionally uncomplicated, but more frequently it is associated with other clinical phenomena, such as hemianopsia, hemianesthesia, and verbal deafness. It has been asserted that word blindness and hemianopsia always occur together, but cases have been reported which disprove this, as, for instance, the one reported by Sérieux which is referred to under motor agraphia. We may have verbal blindness without hemianopsia, and hemianopsia without verbal blindness, although the observations to substantiate this position are few. My views as to cortical visual localization have already been given on pages 342–344. The cortical field of the macula is doubtless also the area wherein are stored the visual images of words or letters. This area is the angulo-occipital region on the lateral surface of the hemisphere, nearly where it was first located by Ferrier. One of the forms of word blindness is that which is due to lesion of this cortical visual area. Alexia, or the inability to write, and visual agraphia, as well as verbal and literal blindness, are produced by lesions limited to this region: although the patients can sometimes write their own names or a few simple words or, in rare cases, a number of words, they apparently do this through touch and the muscular sense.

Pure Word Blindness. Another form of word blindness, sometimes called *pure word blindness*, has been clearly demonstrated by Dejerine and Sérieux. This is the subcortical visual aphasia which has already been defined when speaking of the anatomicopathological varieties of aphasia. It is caused by a lesion which destroys the fibres which pass from both occipital lobes to the left angulo-occipital region. In pure word blindness of this subcortical type, agraphia for spontaneous writing may not be present. The memory pictures can still be revived by the visual centre, and can be made use of by the motor centres for writing. Commonly the lesion producing blindness of this kind also causes hemianopsia, because the adjacent optic radiations of Gratiolet are usually involved. In such a case of pure word blindness, while the patient is able to write spontaneously and from dictation, he is not able to read what he himself has written. He may, however, be able to copy mechanically what he has written, like other sensory aphasics. The patient can understand writing by tracing the letters with his finger. Spontaneous speech is present, as is also the ability to repeat the speech of others, and uttered speech is comprehended.

Literal Blindness. In literal blindness the patient may recognize letters as objects having certain forms; if they are reversed or badly made, he may be aware that something is wrong with them, but he does not know their meaning: he does not know that A is

A, that B is B, or that C is C, etc. Written letters can sometimes be recognized when printed letters cannot. It is probable that this recognition takes place in the graphic motor centre through the muscular sense rather than through the visual sense. The patient has graphic motor memories, which enable him to recall letters even when those which are printed are to him a blank or at least unrecognizable. A patient may suffer from literal blindness and not from verbal blindness proper, or he may be blind to words and not to letters. While this is true, literal blindness is, as a rule, accompanied by verbal blindness. Those who read words by putting the letters of the word together are necessarily blind to words when they lose the memory for letters. That literal blindness may exist without verbal blindness is illustrated by a case reported by Broca. This patient was attacked by partial literal blindness; that is to say, he had lost the visual memories of some letters. He could even read words in which were letters he did not know, and he changed or suppressed letters in words without perceiving what he had done. Such a patient recognizes words as one does a landscape or a face of which he has not analyzed the details. Words are often comprehended by their general form alone. Reading ceases to be phonetic; whatever it was originally, it becomes ideographic. (Brissaud.) In cases both of literal blindness and of verbal blindness the ability to recognize numbers sometimes remains, the numbers being symbols of ideas of a special kind.

Psychic Blindness.—Not infrequently associated with word blindness is another disorder which has been called soul blindness and object blindness. The word apraxia is also used by Kussmaul in practically the same sense, for the loss of the memory of the uses of things, and the understanding of the signs by which things are expressed. In testing for this condition, the physician observes whether the individual examined shows evidences of recognition of objects of various kinds which are presented to him. He may not comprehend the use of the simplest things; and he may also not recognize persons with whom he is intimate. He may recognize another by his voice or by touching him, even when he cannot by sight. A comparable form of mind deafness, or psychic deafness, is observed, in which the patient cannot recognize another by the word used nor by the sound of his voice which should be familiar. The centre for the visual images of things probably includes or is adjacent to that for visual images of words; but both hemicerebrums doubtless take part in the storage of object images, and in nearly equal degree; while for the recognition of words, as for many of the other higher faculties, man is mainly left-brained. A few cases of partial mind blindness are on record in which unilateral lesions were present. Even a form of *psychic blindness for words* has been described. The patient can read the letters and the words, and even

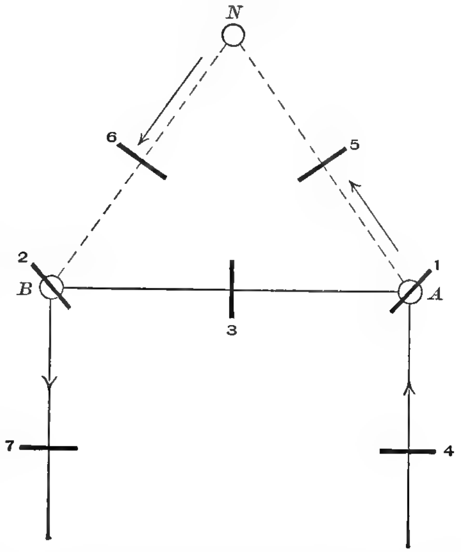
copy them, without getting their sense. Brissaud has compared this psychic blindness for words to the mental condition of a compositor who does not understand Greek and yet is able to set it up correctly and even to make necessary changes and corrections in his work.

Concept Aphasia.—A naming or concept centre has been introduced into the scheme of the cortical mechanism of speech, its probable location being in the third or meditemporal convolution. Some of those who seem to recognize the necessity of a separate centre for names or concepts do not discuss special forms of aphasia dependent upon its lesions. Others differ as to its location and extent. Brissaud places it in the prefrontal region. Broadbent conjectured that it was situated at the under surface of the temporal lobe near its junction with the occipital lobe, and the case recorded by me (page 345) has confirmed this suggestion. In a case of Rosenthal's of verbal amnesia without verbal deafness, an old focus of softening was found in the second and third temporal convolutions, the first temporal having been entirely free from disease. Wyllie holds with many others that it is not necessary to have special "ideational" centres and "naming" centres, but that the interaction of the entire cortex, or of certain of its layers, is concerned with ideation, and that names arise in consciousness through the action of the centres for percepts and the motor centres. Those who do not believe in separate concept or naming centres do not restrict this organization of the cortex to an area absolutely set apart. It seems to me altogether probable, however, that such a region may be located conveniently intermediate between all the receptive and emissive centres concerned in the mechanism of speech. The chief affections of speech and of thought due to lesion of this naming or concept centre are varieties of verbal amnesia or the aphasia of recollection, with usually additional symptoms, such as paraphasia or some variety of sensory or of motor aphasia, because of the frequent and almost necessary involvement of associating tracts. Destruction of this area will cause loss of the memory of names or nouns. A form of verbal amnesia of incomplete type, *incomplete articulative amnesia*, may be due to partial destruction of this region, or of the channels connecting it with its correlated centres on either the receptive or the emissive side of the brain. The *amnesia literarum* to which Wyllie refers may be due to lesion of this concept region. The term is used to indicate "a failure (on the productive side) to call up the images of letters and words in the mind when the effort to write is being made." Instead of regarding concept aphasia as distinct from both sensory and motor aphasia, it is sometimes considered as a variety of sensory aphasia, although the mental process of symbolizing concepts by name is higher than that of perception.

Mixed Concept and Motor Aphasia.—Transcortical motor aphasia is that form of speech disturbance which results from sev-

erance of the path between the naming or concept centre and the centre for motor memories of speech in Broca's convolution. In this form of aphasia the symptoms are similar to those in true cortical motor aphasia, but with some points of difference. According to Lichtheim, the aphasic of this type cannot express his thoughts any better in writing than in volitional speech, although everything else is normal. What he reads and what is said to him are both understood, and he can repeat with correct articulation words that are spoken to him, understanding them. He can also write to dictation and copy writing with ease and correctness, understanding in both cases what he writes. For this mixed variety of speech disturbance Lichtheim has suggested the term outer commissural aphasia.

FIG. 333.



Lichtheim's diagram showing the position of lesions of different tracts and centres producing some of the most important forms of aphasia: *A*, higher auditory centre (centre for word images); *B*, Broca's centre (motor articulatory centre); *N*, concept or naming centre; 1, site of lesion causing word deafness (cortical auditory aphasia, pictorial auditory aphasia); 2, lesion causing motor cortical aphasia (pictorial motor aphasia); 3, lesion causing the conduction aphasia of Wernicke (interpictorial aphasia); 4, lesion causing subcortical auditory aphasia (infrapictorial auditory aphasia, pure word deafness); 5, lesion causing transcortical auditory aphasia (suprapictorial auditory aphasia, inner commissural word deafness, mixed sensory and concept aphasia); 6, lesion causing transcortical motor aphasia (suprapictorial motor aphasia, inner commissural motor aphasia, mixed concept and motor aphasia); 7, lesion causing subcortical motor aphasia (infrapictorial motor aphasia). In order not to complicate the matter too much, several of the important centres and tracts have been omitted in this diagram. By substituting the visual for the auditory centre a series similar to the above would be produced on a background of visual aphasia. The introduction of a graphic centre would give motor cortical agraphia and various forms of conduction, transcortical, and subcortical agraphia.

Motor Aphasias.—*Motor Aphasia of Cortical Origin.* Motor aphasia, whether cortical or subcortical, may be complete or incomplete. When complete, the patient is totally unable to speak, or at most retains only a few simple words. He is unable to repeat words spoken by another, or to read aloud. This loss of power of speaking may exist without any paralysis of the tongue, face, or limbs, although it is commonly associated with hemiplegia or monoplegia. As a rule, it is accompanied by inability to write, this defect constituting *motor agraphia.* When any spoken speech is retained it is of simple much used words, as "yes" or "no," oaths or emotional

ejaculations, or recurring utterances, such as "tan, tan," "la, la," "Oh-no," and "Come-on-to-nong." The motor aphasic understands the meaning of words spoken, and, if sufficiently educated, of words printed and written. He also comprehends the uses and meanings of objects, and can call up their names in consciousness. Close examination, however, shows that his power of calling up auditory images is affected and his ability to read mentally is impaired. He may be able to copy words, or in rare cases, when the power of writing is retained, to translate printed into written words. Compared with other forms of aphasia, pantomime is usually well preserved. The patient may protrude his tongue instinctively or involuntarily, but he is often unable to do this when asked. In many cases of hemiplegia, with motor aphasia total at first, the power of speech is in time largely regained; in others aphasia remains complete until the death of the patient. When recovery, gradual or partial, takes place, simple words and expressions return first. Under the designation "*articulative ataxia*" Wyllie describes what is practically an *incomplete motor aphasia* from partial destruction of Broca's centre. It is simply the old *ataxic aphasia*, the *asynergia verbalis* of Lordat. Such a patient gradually improves in speech through recovery of the injured centre, or through the education of the previously uneducated centre of the right side, and as he does so he shows articulative disturbances of various kinds. In some cases of cortical motor disease, utterance is paralyzed, although the patient does not in reality suffer from an ataxic or asynergic aphasia. The true motor speech centre is not the seat of lesion in these cases, but that area which is marked U in the diagram (Fig. 331) and is sometimes designated as the *utterance centre*. This is the cortical area in which are represented the movements of the throat, larynx, lips, and lower face, those movements which are concerned in phonation, vocalization, and the formation of sounds into words and phrases. For a discussion of the differences in the effects of lesions in this area and in the hinder portion of the left subfrontal convolution, the section on cerebral localization should be consulted at pages 348 and 349. Cases are recorded in which limited destruction of this cortical region has caused impairment or loss of articulate speech, or of the power of utterance, without destroying the patient's ability to recall the psychomotor images which are concerned in speech, while, on the other hand, destruction of the left third frontal makes all speech impossible. In a case recorded by me, one of orolingual monoparesis with distinct paralysis of the lower face, no interference with propositionizing being present, softening involved this utterance centre, the left subfrontal almost entirely escaping.

Subcortical Motor Aphasia. In subcortical or infrapictorial motor aphasia the lesion cuts across only those fibres which connect the

centre for the motor memories of speech in Broca's convolution with the centres for the nerves of speech in the oblongata. Spoken speech, both volitional and on attempted repetition of the words heard, is disabled; there is no amnesia verbalis, no difficulty in volitional writing, writing to dictation, or copying, and no word deafness. If paralysis prevents writing with the right hand, it can still be done with the left.

Motor Agraphia.—Motor agraphia is usually associated with aphasia, hemiplegia, or other clinical phenomena. It may be *complete* or *partial*, but most of the cases associated with aphasia are complete. The motor agraphic retains his full intelligence. He understands what he hears and what he sees of spoken, printed, or written language; he may be able to hold the pencil or pen in proper position in his fingers, and yet be unable to write. He may even be able to copy letters or words in their printed or written forms, or geometrical figures, such as a circle or a triangle. Isolated agraphia is, however, very rare. Some hemiplegics with aphasia and agraphia learn after a time to write with the left hand, and sometimes show a tendency to copy from right to left, and with the letters reversed, this constituting the so-called mirror writing. Motor agraphia is a disorder of coordination rather than a disorder of vision or a true paralysis. The agraphic is not able to coordinate or energize those movements by which ideas are expressed in written signs. In order that a patient shall suffer from true motor agraphia, his writing must be of a specialized character, not merely a translation of his spoken language. It is not rare, as shown by Luys, to observe the dissociation of the faculties of speaking and writing in certain insane persons who are not delirious when they speak, but who are delirious when they write. The reverse may also be witnessed. This proves that the faculty of writing does not consist merely in the power of assembling letters, but in the psychical elaboration of words, phrases, and sentences, of which writing is but the expression. According to some authorities, agraphia is invariably present when cortical motor aphasia is complete; but a few cases opposing this conclusion have been recorded. Brissaud has reported a case of hemiplegia and complete motor aphasia in which the patient was able to write with his partially paralyzed right hand by carefully placing the pencil between the fingers of this hand with the left, with which he also assisted the right hand. A few cases of total motor aphasia have been reported in which the patient could write with the left or unparalyzed hand both spontaneously and from copy. In a case of this kind reported by Kostenitsch, autopsy showed destruction of Broca's convolution. Most cases of motor aphasia are associated with right hemiplegia, and some of these may not be able to write because of the paralysis of the right upper extremity. It was at one time thought that the paralysis was the chief cause of the

agraphia; but cases of pure aphasia without hemiplegia, and others in which the paralysis in the upper extremity is not sufficient to prevent writing, and in which the patient is also agraphic, show that this theory is not correct. It is probable, as stated under cortical localization (pages 349 and 350), that a separate graphic centre exists, and that it is located in the caudal extremity of the medifrontal convolution, in close relation with the mesal boundary of the true speech centres (Fig. 331). This area must not be confounded with that for movements of the fingers, hands, and other parts concerned in writing. The true graphic centre is in advance of this region, just as the true motor speech centre is a little cephalad of the utterance centre; and just as the utterance centre is a region containing motor executive cells through which speech begins to be exteriorized, so that portion of the cortex which is related to the movements concerned in writing may be regarded as the executive cortical region for the graphic centre. Opinion is by no means settled with regard to the existence of this graphic centre. Wyllie concludes that it is probable that the power of writing with the right hand may be lost from a lesion of this centre, but that the left hand would be able to draw the visual images of the letters revived at the visual centre. Dejerine holds that no case of pure and uncomplicated agraphia due to lesion of this centre has been put on record. While recognizing the fact that cases of isolated agraphia are sometimes met with, he believes that in these cases the agraphia is the clinical remnant of an aphasia which has been present and has been caused by lesions either of the sensory or motor centres for speech.

Transcortical Agraphia.—The ability to write is probably lost not only when the higher visual centre is destroyed, but also, at least in part, when the connection of this centre either with Broca's centre or with the graphic centre is interrupted. In one case reported by Pitres, the patient had paresis and rigidity of the right leg, slight paresis of the right arm, and right homonymous hemianopsia. He could copy letters and figures with his right hand, but otherwise was agraphic. He was first seen two years after the apoplectic attack which had caused his symptoms, and in the mean time he had trained himself to write with his left hand. After writing numbers with his left hand he could copy them with his right hand; he had no word blindness, no word deafness, and no difficulty with spoken speech. The existence of the hemianopsia as well as the paralysis showed that this case was not due to an isolated lesion of the graphic centre. The lesion was probably in the centrum ovale involving the motor projection fibres, the optic radiations, and the paths between the visual and the graphic centre or between the concept and the graphic centre.

Conduction Aphasia.—In 1874, Wernicke, under the name of conduction aphasia (*Leitungsaphasie*), described a form of speech

disturbance in which the patient was neither word deaf nor troubled with any form of asynergetic or ataxic speech disturbance, but in which he had marked *paraphasia;* that is, his speech was confused or jargon-like, words or syllables being substituted, transposed, or jumbled together. This form of speech disorder was attributed by Wernicke to lesion of the tracts associating the centre for word hearing and the motor speech centre in the left subfrontal convolution. It is in reality a form of transcortical aphasia, being due to disruption of the tracts associating cortical speech centres. In these cases the lesions have usually been found in the insula and the floor of the Sylvian fossa. As the transcortical aphasias of the mixed sensory and concept types and of the mixed motor and concept types have already been described, it will only be necessary to speak of these cases as distinct when they sever the *direct* pathway between the auditory and motor speech regions. Some of those who recognize the conduction aphasia of Wernicke as a distinctive type do not acknowledge the existence of separate concept and naming centres as taught in this article. Beside the paraphasia or jargon speech, analogous conditions for writing, reading, or pantomime may be present as the result of lesions producing paraphasia. Thus, the patient may suffer from *paragraphia*, his writing being incoherent or disconnected, or from *paralexia*, the words being misused, transposed, or substituted in reading; or he may misuse signs and therefore be a sufferer from *paramimia*. *Dysphasia*, *dysgraphia*, *dyslexia*, and *dysmimia* indicate difficulty or fatigue in articulation, writing, reading, or pantomime, the result of exhaustion or partial lesion of conducting tracts, although these affections may also sometimes be due, in part at least, to exhaustion or partial destruction of centres as well as of tracts.

Amusia and the Aphasia of Intonation.—Theoretically as many varieties of cerebral deafness may exist as there are varieties of auditory symbols. A patient may be musically deaf, for instance, and the deafness may be either a receptive or an emissive amusia. In such cases the adaptation of the conventional sound to the idea it expresses has become impossible. Language does not consist of articulate sounds alone, it is also sung. The faculty of intonation may be preserved even when the motor images of words cannot be revived in the cortex. One of Brissaud's patients, already referred to under motor agraphia, was a woman who was neither word deaf nor word blind. She was able to copy accurately printed or written words, but she was absolutely aphasic so far as the articulation of words was concerned. In attempting to communicate her ideas she not only made use of the facial muscles of expression, but used certain clucking, chattering, gurgling, or shrill sounds, varying the intonations in an infinitely delicate manner. These intonations were modulated like a sort of song, sometimes soft, sometimes loud, sometimes

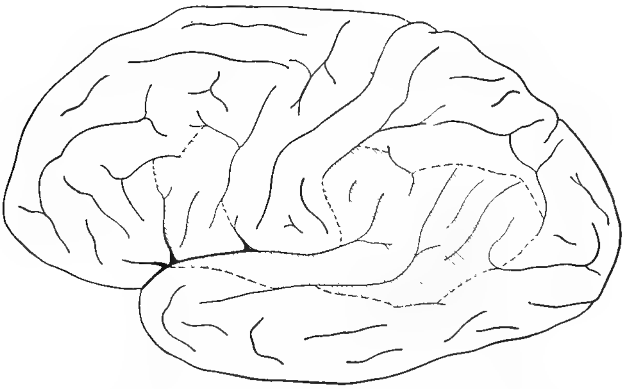
accelerated and sometimes slackened in rhythm, according to the idea she intended to express. The cortical centres for motor images were probably intact. Such a case would seem to prove that the conducting fibres for articulation are not the same as for intonation. Intonation precedes articulation in the development of the language of races as in that of individuals. Articulation is the complement of intonation, not intonation of articulation. Accent, according to Rousseau, is the soul of discourse. Intonation may be used and has been used with great advantage in the training of deaf mutes and imbeciles. It was used by Itard in his attempts to train his famous wild boy. Articulation and intonation are often lost together, but articulation may be lost and intonation remain, as shown by the above case. (Brissaud.) Edren has reported a case of transient paraphasia and verbal deafness, and also permanent deafness for musical notes, in which, at the autopsy, lesions were found in the anterior two-thirds of the first temporal and anterior half of the second temporal lobe of the left hemicerebrum. Fifty-one other cases bearing on the subject were collected by this writer, and of these eleven were with postmortem examination records. He arrives at the following conclusions: (1) Any pathological process within the skull may destroy the musical faculties of the patient in the same way as the speech faculties, thus leading to different clinical forms of amusia; (2) the different forms of amusia have some clinical independence in relation to each other, as well as to the different corresponding forms of aphasia; (3) the clinical forms of amusia show a great analogy with the clinical forms of aphasia, the former being often, not always, accompanied by the corresponding forms of the latter; (4) amusia may occur in the clinical picture without aphasia, and *vice versa*; (5) some of the forms of amusia have very probably an anatomical independence, and are always localized in the neighborhood of the areas of the corresponding forms of aphasia; (6) musical deafness, that is, the perception of notes, accords, and melodies without comprehending their musical meaning, is probably located in the first or the first and second left temporal convolutions, anterior to the area of verbal deafness.

The Nature of Aphasic Phenomena.—*Differing Views.* The statements above made regarding the varieties of aphasia and their symptomatology are, I believe, based upon conclusions which can be justly drawn from clinicopathological observations and a general study of the entire subject. The views taken differ considerably from those of some high authorities, but are largely in accord with those held by others equally high, although differing perhaps in some respects from all. It will serve a good purpose to discuss briefly some of the theoretical points which from time to time have claimed attention, and which are yet the source of controversy not always amiable. If the differing opinions are carefully sifted, they

will be found to be more in agreement than at first sight appears. Much of the seeming difference is due to the manner in which the same phenomena are interpreted by different observers. In not a few cases the differences are matters of words rather than real antagonisms.

The Zone of Language. Special prominence is given by Freud and Dejerine to the idea of a "cerebral zone of language." The extent of this zone is given somewhat differently by different authorities. According to Dejerine, it embraces those portions of the cortex of the left hemicerebrum which are shown in the shaded area in the diagram (Fig. 334). As there indicated, beginning at the ascending branch of the Sylvian fissure, it includes on the motor side the hinder part of the third frontal convolution and the lower extremity of the precentral convolution, extending to but not taking in the second frontal. On the sensory side it is made to include almost the entire

FIG. 334.



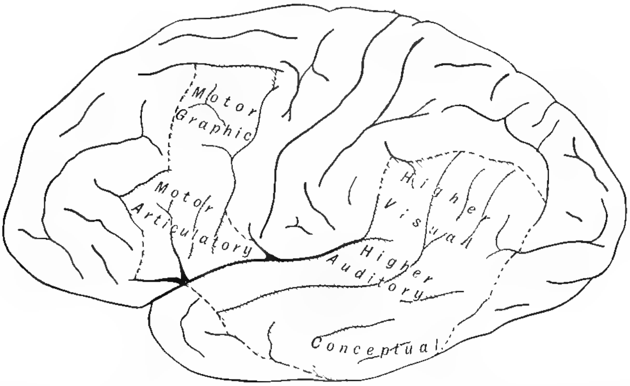
The zone of language according to Dejerine (shown by the shaded area).

first temporal and the posterior extremity of the second temporal convolution, and also those portions of the parietal and occipital lobes which are usually spoken of as the supramarginal and the angular convolution. Its sensory and motor subdivisions are united by way of the Sylvian fossa by means of the associating fibres which pass beneath the insula and the retroinsular convolutions. This zone of language includes three centres,—namely, that for the motor images of articulation, that for auditory images, and that for visual images. Each of these centres is situated in the part of the zone of language which approaches most nearly to its corresponding general zone, motor or sensory. The motor centre of articulation is in proximity to the psychomotor region; the centre of visual images approaches most nearly the general visual zone; and the centre of auditory images is in close relation with the general auditory sphere. In this zone of language the centre for auditory images is that first

evolved and most deeply organized. As a rule, the child's first ideas of language come through hearing; articulate speech is next evolved; the child hears, and it speaks; it learns to repeat the names of persons and objects with which it comes into relation; later, in those who become educated, a centre for the visual images of letters and words is organized, and still later, at least according to some authorities, a centre for graphic motor images. The auditory and motor speech centres continue to be for the vast majority of people most important constituents of the zone of language. According to Dejerine, alteration of any one of his three centres for sensory or motor images causes a change in language which implicates the functions of all parts of the zone of language; alterations in the tracts which associate these centres lead also to some change in all the forms or modes of language. Certain symptoms predominate, however, according to the particular centre or tract directly and most profoundly affected. If Dejerine's visual centre is destroyed, the functions of Wernicke's auditory centre and even, to a less degree, those of Broca's motor centre are impaired. If Broca's centre is destroyed, the functions of the visual and auditory sensory centres are affected. If Wernicke's centre is destroyed, those of the visual and articulatory centres and all parts of the zone of language are perverted. Motor aphasia dominates when the lesion is situated in the centre of Broca, verbal deafness when in Wernicke's centre, and verbal blindness when in the angular convolution, but in each and every case, according to the view of Dejerine, all other modes of language are altered. Verbal blindness, either manifest or latent, is always present in cortical motor aphasia; and while verbal deafness, as a rule, is not present, or at least does not continue, the power of spontaneously evoking auditory images is impaired. It is held by the advocates of this view that the facts which prove the involvement of all parts of the zone of language are frequently overlooked owing to the carelessness, or at least to the want of precision and fulness, in the methods of examining patients. While for many years the study of motor aphasia has been a favorite one and has not infrequently been conducted along somewhat elaborate lines, it is only recently that its investigation has been made sufficiently methodical and complete to give results of much value regarding disputed questions. It will be seen that the conception of aphasia and of a zone of language just given is not entirely in accord with the views indicated in the diagrams Figs. 331 and 332 and with the teachings of the preceding portions of this section. I would enlarge the zone of language, as given by Dejerine, so as to make it include a centre for concepts in the third temporal convolution and possibly extending over more of the midtemporal region, and in addition a graphic motor centre in the caudal portion of the second frontal convolution (Fig. 335). This zone of language unquestionably has its deepest organization and highest

development in the region encircling the Sylvian fossa, for here are situated the auditory centre out of which the others may be said to have been evolved, and the motor articulatory and visual centres which are next in importance as they have been next in development; but it must also include those portions of the brain in which concepts originate, and, if the views of those who believe in separate graphic motor centres are correct, also those parts in which graphic motor images are represented. Moreover, language on its productive side may result from impressions received through any of the sensory gateways; written and spoken language is evoked not only by auditory and visual images, but also through olfactory, tactile, gustatory, and spatial percepts; so that in a lesser degree the centres for touch, taste, smell, and other senses than those of audition and

Fig. 335.



The zone of language according to the author. This is made to include the midtemporal region as the probable location of the area for concepts, and also the caudal portion of the second frontal convolution as the graphic motor centre. The motor articulatory centre is made to extend a slight distance in front of the ascending branch of the Sylvian fissure, which is in accordance with the author's general diagram of the cortical centres and areas. (See Fig. 228.)

vision, and the tracts which associate these centres with the motor side of the brain, may be regarded as properly constituting, at least at times, a part of the zone of language.

The Separate Existence of Concept Centres.—Let us again briefly direct our attention especially to the question of concept centres or areas, and to the part which they play in the mechanism of speech. Even Lichtheim, who finds it necessary to make use of concept centres in his schemes, does not regard such centres as having a local or at least a restricted habitation. In his diagrams he has represented a centre for concepts, but this he says he has done for simplicity's sake, believing with others that the function of forming concepts is not localized in any one spot of the brain, but results rather from the combined action of the whole sensorial sphere. According to him, the concept centre should be distributed over

many spots, radiations from which converge to the sensory and motor speech centres. He holds that in order to have all the commissural connections broken between the concept centre or centres and either the sensory or the motor centres for speech, the lesion should be close to the percept centres on the one hand or to the motor centres on the other. The matter does not present itself to me in exactly this way. Structural and functional lines pass from all the centres on the sensory side, to reach eventually by direct or indirect paths the centre of Broca. The locations of the separate centres are such that the structural connections are most probably made by way of the midbasal portions of the temporal lobe. The recognized sensory percept centres surround this region, and within it fibres from them to the motor areas probably converge and cross. As stated when discussing the naming centre, Broadbent long ago conjectured that this would naturally be the location of such a centre. This "centre" need not be as strictly limited anatomically as the separate centres for percepts, but it would occupy a region more or less localized, although it might be diffused over a considerable area. That differences of opinion about aphasic phenomena are sometimes apparent rather than real is illustrated by this discussion of the question of the existence of a separate concept centre. Freud contends that aphasia is always caused by disruption of associating tracts or paths, and that different forms of aphasia occur with certain symptoms predominating according to the position in his zone of language in which such disruption takes place. If a destructive lesion attacks the centre of his zone, the disorder of language will be such as to involve all the forms and phases of language. The centre of such a system of associations would be, anatomically speaking, where the structural lines which pass from the different sensory to the motor centres of speech cross, commingle, or most nearly approach one another. The symptoms presented when the midtemporal region is destroyed, as illustrated by my naming centre case, indicate interference with all the modalities or elements of language. The verbal amnesia, or the aphasia of recollection, caused by lesion of this region, includes within it some disturbance of all the elements of speech. In a typical case the patient cannot recall names through sight, touch, hearing, smell, or any other sense, and as they cannot be recalled they cannot be written or spoken, at least not spontaneously. The patient may repeat after another, may copy or may write to dictation, but he does this by using the direct pathways between the sensory and the motor centres. He understands the meanings and the uses of the objects for which the names stand because his percept centres are intact. Ross held that in passing from thinking by percepts to thinking by concepts, and from that to thinking by abstracts, no new centres are introduced, but only complication upon complication of the percept centre or centres.

The evolution of such a complex mechanism, however, necessitates the enlargement of the sensory percept areas, and this would have as its particular result the development of a new structural area intercalated between the receptive and the emissive portions of the brain, but more closely related to the former than to the latter. When lesions occur near, but not in, the sensory inlets, a disorder of language, conceptual in character, results. The portions of the cortex the activity of which is related to thinking by concepts and abstracts can be reached in the word blind through the eye, in the word deaf through the ear, and in those who cerebrally are both deaf and blind through the nerves of the other special senses. If this be so, in some region of the brain the structural lines must converge or at least commingle and cross on their way from the sensory centres to the motor centres, and this area, however restricted or extended, constitutes the concept region of the zone of language. The symptomatology which results from a lesion of this concept area is that which was present in the case reported by the writer as localizing this naming or concept centre. Somewhat similar cases are sometimes spoken of as cases of optic aphasia, but our case was more than this; the patient was unable to recall through either the sense of sight or that of touch the names of objects the uses of which she evidently recognized. A typically perfect case would be one in which the patient should be unable to call up through any of the senses the name of an object the use and meaning of which are comprehended.

The Question of a Motor Graphic Centre.—In the paragraph on motor agraphia I have indicated my belief in the existence of a motor graphic centre and the possibility of having a pure motor agraphia from lesion of this centre. To some extent I have discussed the subject; but so much that relates to the whole subject of aphasia, and especially to that of interior language, is involved in the question of agraphia that it may be well to consider further the views held by opposing authorities. In the days of Trousseau, and later, motor agraphia was generally regarded as dependent upon motor aphasia. The doctrine of a distinct graphic centre, however, soon received considerable support, and for years was largely accepted; but recently the tendency has been to return to the view of Trousseau. Déjerine in particular strongly upholds this view, which has been presented by him in special articles, and is ably discussed by his disciple Mirallié in his valuable monograph on sensorial aphasia. Wernicke, Lichtheim, Sérieux, and many others in recent years have acceded to this view. On the whole, however, the facts are in favor of the existence of a separate graphic motor centre. Arguments psychological, physiological, clinical, and pathological have been marshalled for both sides of the discussion. The psychological arguments in favor of the existence of such a centre were best stated by Charcot and have

been best defended by Ballet. According to them, a word is the result of the association of four distinct images,—auditory, visual, articulatory, and graphic. During silent reflection we hear, see, or speak mentally, and in special instances we think by means of graphic images. When we think about a word or words, the first images revived are those which constitute the primary couple in language,—namely, the auditory and motor articulatory; when we read, the visual images are revived, and along with them also the auditory and motor articulatory images; in writing, the visual images are revived in those who can see, and in addition the graphic motor images. When writing is a medium for the expression of thought, all four images are revived, in different degrees in different individuals according to their mental aptness and special training. In some, auditory and motor images, in others the visual, and in others, probably few in number, the graphic images, play the most important part. The recorded clinicopathological observations in support of and in opposition to this view seem to me to be of almost equal weight. On the one hand it is held that nearly all cases of so-called motor aphasia have agraphia associated with them; that in visual and even in auditory aphasias some degree of agraphia is usually present; that cases of agraphia unassociated with visual, auditory, or motor articulatory defect are almost unknown; and that when agraphia is present without aphasia it is usually the relic or remnant of a previous aphasia. On the other hand, it is contended that a few clinical cases with autopsies support the conception of the existence of a separate motor graphic centre; that in rare cases agraphia has been absent when motor aphasia has been almost total; that agraphia is sometimes observed when neither auditory nor visual defect is present, and that in exceedingly rare cases agraphia has been noted as the result of an isolated lesion. Certainly the well-known cases recorded by Charcot, Bar, Kostenitsch, Ogle, and others lend considerable support to the view that favors the existence of a separate centre. In Bar's case a lesion was found limited strictly to the second frontal convolution, the patient being a total agraphic and also an aphasic; here the third frontal convolution escaped, and the agraphia was due to a lesion of the second frontal. It is probable that the aphasia was dependent upon the pressure exerted upon the neighboring third frontal or upon the destruction of some of its subcortical or association fibres. In Kostenitsch's case the third frontal cortex as well as subcortex was certainly destroyed in very large part, and yet the patient was not agraphic. Against the doctrine of a separate graphic centre has been arrayed the fact that certain individuals learn to write with their toes, elbow, knee, or mouth; but this argument does not seem to me to have as much weight as is usually given to it. If a graphic centre exists, it is distinct from the centre for movements of the finger and hand, just as the true motor speech centre is distinct from the centres for the move-

ments of the lips, tongue, and face. When writing is performed by unusual methods, the graphic motor images are revived in the graphic motor centre, but they are exteriorized by different portions of the so-called motor zone, associations being formed between the graphic motor centre and the different regions for motor exteriorization. The same thought is applicable to the explanation of writing with the left hand instead of with the right. It has been found that aphasic patients are awkward in forming letters or words with alphabetical cubes or blocks just in proportion to the degree of aphasia and agraphia from which they suffer, and hence it is argued that a graphic motor centre does not exist, and that the defects shown are due not to loss of graphic images, but to the loss of the notion of the word or words. With equal force it might be argued that it is not the loss of articulatory images but the loss of the notion of the word which causes the aphasia, the notion of the word not being dependent, entirely at least, upon either graphic or motor articulatory images, but upon the associated action of the entire zone of language. The fact that the congenitally blind, who have no visual images, can be trained to write or print their names in ordinary characters is in favor of the existence of a separate motor graphic centre. Having no visual images, they must depend exclusively upon the motor memories of writing, what some authorities term the kinesthetic memories of writing. (Wyllie.) The argument would seem to be still stronger in the case of those who are both blind and deaf and who have been taught to write and to give expression to thought in writing. Recently in the nervous wards of the Philadelphia Hospital, in the service of Dr. James Hendrie Lloyd, I have had the opportunity of studying with him and with Dr. W. G. Spiller a patient whose symptoms seem to bear out the idea of the existence of a separate graphic centre. This patient has disturbances of language which have been of gradual development. He is not word deaf, he is not a true motor aphasic, but he is a total agraphic. He is only able to copy mechanically printed letters or words. At times he seems to have great difficulty in recognizing words or special letters; so that it is difficult to decide whether he is suffering from literal or word blindness. The ophthalmoscope shows considerable optic atrophy, and this may be partly responsible for his visual defects in reading. He is not paralyzed in either the upper or the lower extremities, but has slight paresis of the right face. His condition varies; on some occasions he can talk with comparative fluency, but never perfectly; at other times he has moderate or great difficulty either in remembering or in articulating words, it is difficult to say which. He has an area of extreme tenderness which corresponds externally to the first and second left frontal convolutions, and in this area frequent tests seem to indicate the presence of percussion dulness. The case is somewhat perplexing, but it is probable that a tumor or other lesion involves the first two

frontal convolutions. It is not improbable that the patient is also suffering from chronic arterial sclerosis with softening and recurring edemas. Albumen and casts are present in his urine.

Relative Importance of Centres and Association Tracts.—Usually most importance is given to "centres" in the explanation of the phenomena and affections of speech; but Freud lays greatest stress upon the part played by association fibres, and, according to him, lesions of the centres are to be relegated to the second place. Wernicke, Lichtheim, and others have paid especial attention to the aphasias of conduction,—affections due to disruptions of association tracts. It has been shown that their lesions give rise to special morbid phenomena, but their importance is not greater than that which is assigned to lesions of the centres for images. These so-called conduction aphasias have not been regarded as playing even an equal rôle with the disorders due to lesions of the various centres. The forms of subcortical and transcortical aphasia described are, from the standpoint of most observers, restricted to lesions of association tracts. The usual conception is that all forms of aphasia are due to isolated lesions either of centres or tracts or of both in the same case; but with Freud, lesions of the association fibres, which cause disruption of the functions which take part in the mechanism of speech, are of dominating importance in the production of aphasias. For him the aphasic symptom or syndrome is above all a sign of dissociation, speech being altered because the patient is not able mentally to pass from one image to another. According to the extent and degree of the destruction of associations is the impairment of speech. The so-called centres, placed in the periphery of the zone of language, are only points farthest removed from other centres, where therefore the fibres of association are least abundant and where a localized lesion is more likely to include only a single element of language. A destructive lesion at the place where the fibres of association cross among themselves will give for the clinical picture alterations in all the modes in which language is manifested. Considerable credit is due to Freud for directing the attention of neurologists to the importance of association tracts in the pathology of aphasia; but mistakes are likely to be made as regards both centres and association systems. As stated elsewhere, the use of the term centre in neurology, while important, is largely a matter of convenience. A centre is simply a collection of gray and white matter which represents physiologically some action, function, or faculty. Its destruction causes loss or impairment of function, and its irritation leads to excess of functional activity. Centres, like cells, are too often considered as points where force originates. They are best regarded simply as points in reflex arcs or in systems of association, interference with which causes definite phenomena. Impulses, we now know, are transmitted from nerve cell to nerve

cell by contact, and not by continuous fibres over distances indefinitely prolonged. It is an error to make too great a distinction between the effects of lesions of association tracts and of centres. Centre and tract each plays its own important rôle, and it is for us simply to learn, through clinical and pathological observation, the results caused by their lesions when these are of definite size and in definite locations.

Importance of the Distinction between Pure Aphasias and Aphasias due to Impairment of the Mechanism of Interior Language.—The distinction between the so-called pure aphasias, which include pure motor aphasia (subcortical motor aphasia), pure verbal deafness (subcortical auditory aphasia), and pure verbal blindness (subcortical visual aphasia), and the aphasias due to disturbances of interior language as the result of lesions of the so-called zone of language, is a general one which must prove helpful to the physician in the differential diagnosis of the cerebral affections of speech. Word deafness or word blindness as the result of isolated lesion of the tracts which pass from the primary cortical centres for vision and audition to the higher secondary centres for word vision and word hearing is not associated with other disturbances of language, such as verbal amnesia, motor aphasia, agraphia, or paraphasia. The patient's interior language remains intact. Similarly the patient who is suffering from the pure form of motor aphasia, from defect of speech due to destruction of the subcortical projection fibres, has a pure disorder of motor speech. Articulation, and it may be enunciation, are greatly impaired, but he does not in any degree, if the affection is complete, suffer from word blindness, word deafness, dyslexia, alexia, paraphasia, or even agraphia, although he may have an apparent agraphia because of the paralysis of the upper extremity which usually accompanies the aphasia. It is rare, however, to meet with an entirely pure case of either of the forms of sensory or motor aphasia, as the destruction produced by disease does not commonly confine itself strictly to special centres and tracts.

Disorders of Pantomime among Aphasics.—*Definition of Pantomime, and Varieties of Pantomimic Disorder.* Pantomime is the representation of thoughts or ideas by action or movements, and, like the higher forms of speech, is a method of intellectual intercourse. The individual by pantomime may show that he hears, sees, or otherwise receives impressions, or that he is conscious of what is meant by the words or actions of another. He may indicate that certain thoughts have arisen spontaneously in his mind, or he may make use of this method of expression to convey the ideas to others. Pantomime, like speech, may therefore have its sensory or receptive aspect, its concept sphere, and its motor or emissive side. Disorders of pantomime and of speech may go hand in hand, or may be more or

less dissociated; in some cases they may be entirely independent of one another. The rule is that cerebral disturbances of speech have accompanying pantomimic disorders. These are forms of *asemia*, which is the inability to understand, form, or express any symbol of thought or of feeling. In a study of cases a distinction must be made between pantomime and gesticulation; the former is a higher process than the latter. According to Hughlings Jackson, pantomime differs from gesticulation as a proposition differs from an oath. The former is a method of presenting ideas, the latter of giving vent to feelings. The same action, however, may be pantomime in one and in another gesticulation, as when movements of the head or limbs are used in one case to make clear a meaning and in another to exhibit emotion or mere emphasis. The chief varieties of pantomimic disorder observed among aphasics are *amimia* and *paramimia*. Amimia, which is a loss of the power of expressing thoughts by signs or movements, may be sensory, conceptual, motor, or mixed. It may also have special varieties, as *musical amimia*, in which the patient has lost the power of receiving or interpreting music by means of signs or movements. He may be unable to play upon instruments, although he has been a thorough musician and is still capable of appreciating the music. Paramimia, a somewhat common disorder among aphasics, is an affection in which the patient misuses, transposes, or confuses signs intended to convey his meaning. It is a disorder which, as regards signs, is comparable to paraphasia for speech, paralexia for reading, and paragraphia for writing. It may be due to lesions of either centres or tracts, or of both in the same case. It is most frequently dependent upon the destruction of the tracts which associate the sensory, concept, and motor centres.

Relation of Pantomime to the Varieties of Aphasia. Attention will be directed to a few points in connection with the relation of pantomime to the different varieties of aphasia and to its diagnostic importance. I have made a study of pantomime in a large number of cases of different varieties of aphasia. In cases of word deafness or word blindness pantomime is most disturbed on the sensory or receptive side. If these disorders are associated with verbal amnesia and any of the forms of apraxia, marked disturbances of pantomime will be present. In true motor aphasia, even when this is complete or nearly complete, the patient sometimes shows marked preservation of pantomime. Such aphasia is, of course, usually accompanied by right hemiplegia, but the patients have the full use of the left upper extremity, as well as of the head and other parts of the body, and with these are often able to communicate by pantomime. In some cases, however, of even ordinary motor aphasia, pantomime is more or less impaired, the difference being probably dependent in part at least upon the ability of different individuals to use both sides of their body in signs and pantomime. In mixed sensorimotor aphasia

pantomime may be of very uncertain character. One marked case of this kind was examined by me, with the result of showing that her pantomime, which was usually correct, had in it an element of uncertainty, slowness, and awkwardness. (Fig. 336.) She pointed, beckoned, and motioned away, but awkwardly. On being told to close her eyes, she did so promptly; on being told to lift her right hand, she tried to raise this which was her paralyzed hand, and, finding she could not do it, said, "Can't tell." When asked whether

Fig. 336.

Right hemiplegia with marked contractures; almost complete aphasia, with unreliable and confusing pantomime; patient fifty-four years old; she had had two attacks of apoplexy twenty-three years before observation; lesion probably subcortical and capsular.

she was married, she said, "Yes," with the proper forward nod of assent, but immediately after said, "No," shaking her head properly for dissent. On being asked if she had headache, she said, "Yes," with an assenting nod. When asked if she had any houses, she nodded, "Yes," and assented when she was asked if she had but one house; she responded similarly to various tests. She could assent and dissent by pantomime understandingly: she did not, however, always do this correctly, but usually she corrected herself when she was wrong.

Cases with Limited Speech and Limited Pantomime.—Cases which retain only a single recurring utterance are usually almost equally limited in pantomime. Just as such cases have only one or a few utterances, so they may have only one or a few recurring signs, as an energetic movement of one arm, or a movement of the head accompanied by a certain facial expression, as shown in Fig. 337. This patient's only utterance is, "La, la," and the case is one of almost complete amimia. When she makes use of this recurring utterance, it is almost invariably accompanied by the facial expression which has been caught in the photograph. Subcortical motor aphasics preserve the power of pantomime. Pantomime is also largely preserved in subcortical sensory aphasia, although in subcortical visual aphasia the patient may in some instances be blind to signs as he is to letters. The "yes" and "no" of an aphasic are well known to have very diverse degrees of value. One of these two words may be used to express both assent and dissent; or with its proper meaning; or to express assent when dissent is meant; or simply as an emotional or an accidental expression. In like manner the usual pantomimic methods of expressing assent by the forward nod or bowing of the head, and of dissent by shakes or half rotations of the head, will be found in aphasics to have as many interpretations as the articulated "yes" and "no."

Fig. 337.

Right hemiplegia with marked contractures; complete aphasia of the mixed type; a single recurring utterance and recurring facial expression; almost complete amimia.

Diagnostic Points regarding Pantomime. Great care should be taken not to misinterpret the emotional manifestations of an aphasic. The gestures and appearance of the face indicative of displeasure, anger, obstinacy, or irritability are often strongly suggestive of dissent; while, on the other hand, those which merely indicate pleasure, amusement, or playfulness may sometimes be mistaken for assent or accord. When the lesion is entirely in the straits between the ganglia, the corona radiata escaping, pantomime is either not lost or is soon regained. The speech defect in such a case is of the nature of an *anarthria*,—that is, a defect or disturbance of articulation. It is a pseudobulbar affection. A diagnostic point is the ability of the patient to throw even into the paralyzed mem-

bers some volition. In numerous instances hemiplegic patients who at first suffer from marked paralysis of the leg, arm, and face, and complete or nearly complete aphasia, recover largely control over the face, and ability to speak as well as to use pantomime, while loss of power in the leg and arm remains. This form of hemiplegia is familiar to all neurologists. In patients of this class the lesion is usually situated in the posterior half of the internal capsule, involving often the lenticular body.

Speech Disturbances and Mental Disease.—From lesion of the prefrontal lobes or from general deterioration of the brain a patient may suffer from *alogia*, or inability to speak owing to defect of his higher psychical faculties, and this may be dependent upon a diffuse destructive cerebral lesion. In various forms of idiocy and imbecility, absence of speech may not be in a strict sense of an aphasic character, but rather dependent upon the general lack of intelligence, being one of numerous evidences of arrested mental development. In some forms of delusional insanity the patients are prevented from speaking by their delusions. Aphasia dissociated from marked mental impairment is of more frequent occurrence than aphasia in association with such impairment; so that in a case of suspected insanity the burden of proof rests on those who maintain that mental disease is present. "The varieties of insanity which are most apt to be complicated by the occurrence of functional aphasia are those in which there is obvious depression of the nervous energies of the brain. The depression of energy may be lasting, as in senile dementia and acute melancholia, or it may be temporary, as after one or other of the numerous forms of nervous seizure which the insane are peculiarly apt to experience from time to time." (Wyllie.) Amnesic aphasia is often one of the most striking phenomena of senile dementia, and it may or may not be associated with disorders of articulation. In melancholia, as Seglas has shown, not only is mutism sometimes present, but the patient may exhibit what may be regarded as word deafness due to functional exhaustion or disablement. He hears sounds, but he does not recognize them as words. Occasionally an aphasic is supposed to be demented or deranged when he is simply disturbed in mind, he being apparently incapacitated mentally because he is deprived of his ability to communicate with others by language or signs or both. Cases of this kind have evident medico-legal importance. Hallucinations of hearing, of sight, of speech, and the various forms of mixed hallucinatory disorders from which the insane suffer, are doubtless frequently due to functional or organic irritation of the various cortical centres and tracts concerned in speech phenomena. In a disease like general paralysis of the insane, with a well known meningeal and cortical pathology, the speech defects have a real organic basis. It is unusual to observe a case of complete sensory,

motor, or mixed aphasia in paretics or pseudoparetics, but occasionally they are seen as the result of intercurrent apoplexy or of the invasion of a syphilitic organic lesion. In paralytic dementia a combination of active pathological conditions and secondary degenerative changes has sapped the integrity of some or, it may be, of all parts of the cortex required in the mechanism of normal speech, so that we may have any variety, usually incomplete, of paralytic or ataxic speech, or of speech defects conditioned by changes which have taken place in receptive or conceptive cortical areas. In senile dementia the cerebral cortex is more or less deteriorated and disintegrated, and hence, among other phenomena, we have the uncertain, tremulous, mumbling, and piping speech of the aged dement. Pick has recorded a case of senile atrophy presenting the symptoms of transcortical sensory aphasia. The patient had lost the understanding of vocal and written speech, was paraphasic, and had partial loss of ability to repeat spoken words. W. Bevan Lewis has reported a similar case, cited by Pick, in which the patient presented amnesic and ataxic aphasia (mixed sensory and motor aphasia), and in which the brain showed marked atrophy of the left frontal and parietal lobes and less pronounced general atrophy. A few other similar cases have been reported. Seglas, Robertson, and Wyllie have paid special attention to the forms of speech disorder among the insane. The activity of the various centres or tracts concerned in speech, or at least of some of them, is suppressed or inhibited in melancholia, is increased in mania, and may be perverted in various ways in both of these mental affections. The hallucinations and delusions in progressive systematized insanity are dependent upon, or are concomitants of, localized cortical perturbation.

Mirror Writing.—*Definition and General Remarks.* Mirror writing is so called because it can be easily read in a mirror, in which the reflection appears as ordinary writing. Erlenmeyer, in a monograph published in 1879, called particular attention to this curious defect, and it has received attention from a few investigators, particularly those interested in the study of brain physiology in its relations to disorders of speech and of writing, and those who have discussed the dual action of the brain. According to Savage, mirror writing is met with in some forms of mental weakness and in conditions of mental disorder allied to hysteria; occurring also in cases of moral perversion, where it may be only temporary. It is observed more commonly among women than among men, and is most easily acquired in highly nervous people. In writing or copying a word from right to left, one would ordinarily trace first the last letter, and then the next, and so on backward to the beginning of the word. In mirror writing the image of the word is reversed, the first letter in it appearing on the right, so that the word will appear in the ordinary position when reflected from the mirror or when seen from

behind through transparent or translucent paper. Mirror writing can, of course, be done as a trick, or for amusement, and with the right or the left hand, but those who try it will usually at first find it difficult to make particular letters, and especially to connect them consecutively. Dr. Wilbur sent to Dr. Ireland specimens of the writing of a man who could write the same words with both hands at once, the writing done with the left being mirror writing; but, as he could do the same with both hands moving from left to right in ordinary text, the performance was probably sleight of hand.

Illustrative Cases. Ireland gives the details of several interesting cases. One was a paralytic, imbecile girl, between eleven and twelve years old; another was a genetous imbecile girl of fourteen; another, a congenital imbecile of twelve; another, a boy of thirteen of moderate intelligence: all of these defective children wrote with their left hands. He also mentions two left-handed idiot boys who formed pot-hooks from right to left. Buchwald, cited by Ireland, has reported the case of a right hemiplegic and aphasic, forty-five years old, who soon learned to write in a skilful manner, but in mirror writing, his name, as well as the numerals from one to ten, except the figure eight, which he had forgotten. Although the reverse direction of his writing was pointed out to him, he could not be induced to try writing from left to right; and although by persistence he learned to copy some things correctly but awkwardly, he would again fall into the mirror writing.

Mirror Writing and Left-handedness. The left-handed show a physiological tendency to mirror writing. Of a class of sixty boys and girls who tried to write their names with their left hands, two boys and three girls wrote in mirror writing, and all of these were found to be left-handed. Out of another set of one hundred and thirty-four children, six were left-handed, and three of these were mirror writers. Imbecile children who are often left-handed also frequently show a tendency to reversions in spelling and in pronouncing words. One of Leonardo da Vinci's manuscripts is an example of mirror writing, and it has been supposed that this singular style was adopted to preserve the work from superficial readers; but another reason is suggested. A priest who visited Leonardo during the last years of his life has recorded the fact that he had paralysis of the right hand, and, as he was unable to use his right hand, it may be that he learned to write with his left and became a mirror writer. A telegraph operator has informed me that at times when an operator is occupied in sending a message with the right hand, and wishes without stopping to make a memorandum with the left, often the letters are reversed, B, for instance, being written ᗺ. Two explanations of this are suggested: one, that the operator can with more readiness write in the centrifugal direction from the trunk, and will therefore incline to reverse the letters;

the other, that the left hemisphere being intensely occupied with both the mental and the manual effort required in sending a message, the right takes separate charge of the left hand in making the memorandum.

Mechanism and Pathology of Mirror Writing. Ireland comments as follows on the probable mechanism of mirror writing: "It may be asked, is the image or impression, or change in the brain tissue from which the image is formed in the mind of the mirror writer, reversed like the negative of a photograph; or if a double vision be formed in the visual centre, one in the right hemisphere of the brain and the other in the left, do the images lie to each other in the opposite directions, *e.g.*, C on the right side and Ɔ on the left side? We can thus conceive that the image on the left side of the brain being effaced through disease, the inverse image would remain in the right hemisphere, which would render the patient apt to trace letters from right to left, the execution of which would be rendered all the more natural from the greater facility of the left hand to work in a centrifugal direction. Moreover, when one used the left hand to write, there would be probably a tendency to copy the inverse impression or image on the right side of the brain." Special convolutions in the right hemisphere have in a quiescent and undeveloped state the same functions that are active in the corresponding convolutions of the left hemisphere, as many observations on aphasic patients have demonstrated. The right side of the brain is not functionless, but its functions are in part, and sometimes largely, in abeyance. When the individual has the perfect use of the left half of his brain,—is right-handed, has the usual expertness with his right hand, has speech and vision in accordance with his inheritance and his training,—impressions which come to him through his eyes are received by both the lower and the higher visual centres, so as to present to his consciousness a normal or a usual image, which, as a rule, has been registered only by the visual centre of the left hemisphere. In this centre the images are recognized as in correct position; the image formed on the right side, if one exists, is probably usually suppressed. When now the left side of the brain is destroyed, or when its development has been arrested, the individual is guided in writing by images formed in the right side of the brain. In Fig. 338 is given the mirror writing of the patient shown in Fig. 330 on page 614.

Pathological Etiology and Etiological Diagnosis of Aphasia.—The pathological cause of aphasia may be hemorrhage, softening, meningitis, encephalitis, tumor, or almost any organic cerebral affection which may also give rise to loss, impairment, or disturbances of speech which strictly are not aphasias. The distinction between aphasia and the various hysterical affections of speech, such as aphonia, hysterical mutism, hysterical stammering, etc., is an in-

teresting practical matter, but is best considered in connection with hysteria. One or two diagnostic points may here be noted. A case of hysterical mutism or aphonia usually retains the power of writing, while the true aphasic, as a rule, is also agraphic; but it must be remembered that good authorities, as Ladame, believe that an affection which is properly designated aphasia and may be associated with agraphia is sometimes found among the hysterical. In catalepsy, astasia abasia, and chorea, marked disorders of speech are often present. The diagnosis of their real nature is to be made by a close scrutiny of the affection in which they occur, and by a comparison of the speech phenomena with those of true aphasia. The disturbances of speech observed in catalepsy and astasia abasia are hysterical in character, like the other phenomena of these affections. In chorea they are usually dependent upon incoordination of the muscles

FIG. 338.

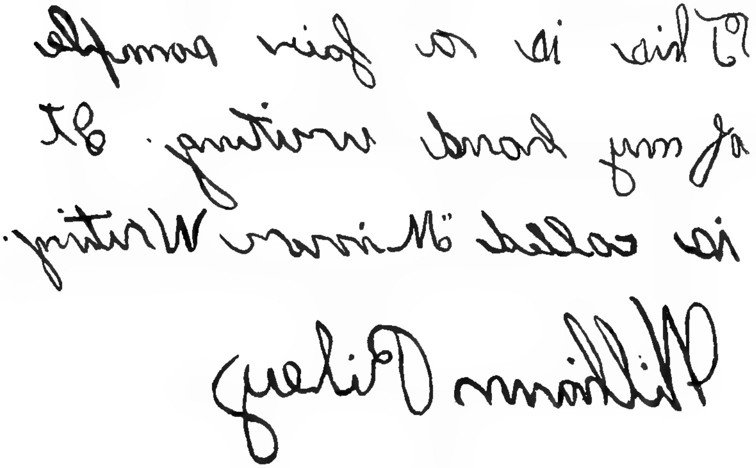


Illustration of mirror writing.

concerned in articulation, phonation, and respiration; although a true amnesic aphasia may sometimes be exhibited. Occasionally aphasia and other disturbances of speech are due to reflex irritation; among cases of this kind which have been put on record are those due to worms or to accumulations of scybala or other irritating matter in the intestines. The occurrence of aphasia and other troubles of speech as the result of toxemia needs to be borne in mind. Any toxic or infectious substance which acts powerfully upon the brain may cause aphasia. Among the substances introduced from without which are known to have acted in this way are tobacco, belladonna, opium, cannabis indica, alcohol, the poison of snakes, and lead. Among the toxic agents formed within the body are those which are present in the uremia of nephritis and the acetonemia of diabetes; also those which occur in typhoid and typhus

fever, variola, measles, and malarial fevers; in fact, any of the infectious diseases may cause aphasia, and, as has already been shown when considering the cerebral palsies of children, may lead to paralysis. A distinction must be made between cases of temporary aphasia, in which the speech disorder is simply a nervous symptom due perhaps to anemia and exhaustion of the brain, and those cases in which hemorrhage or embolism occurs during the course of an infectious fever and gives rise to hemiplegia, monoplegia, or aphasia, or to a combination of aphasia with one of the forms of paralysis. A form of *dysarthria* or difficulty of articulation which occurs during fevers has been particularly studied by Westphal, who describes a slow staccato utterance like that of multiple sclerosis as not infrequent in the course of smallpox. Slurring of speech when extreme is a grave symptom of fevers. (Wyllie.)

Systematic Examination of Patients for Aphasia.—In order to determine that aphasia is present, and its variety, the examination of a patient should be thorough and systematic. It is best in a general way to follow methods which are suggested by the order in which the different forms of aphasia have been considered, although for special reasons this order may be departed from and the patient tested for whatever seems the most important at the time. The patient should be examined critically for word deafness, and in doing this it may be necessary to determine first that he is not deaf for ordinary sounds, and that his deafness is not due to peripheral disease. It should also be determined whether or not he can read; and if he can read aloud, whether he does so correctly or whether his reading is of a stumbling kind,—that is, whether he suffers from paralexia. He should be studied for word blindness. If he suffers from verbal or literal blindness or from both, further examination should be made to determine whether he can write spontaneously and from copy or from dictation. The manner in which he writes or copies should be particularly observed. It should be ascertained whether he understands what he writes, and also whether he can read what he himself has written. It must, of course, be determined whether his defects in writing are due to ignorance, want of education, or peripheral defects in vision, or to inability to coordinate and synergize the movements necessary in writing, instead of being dependent upon word or letter blindness. Wyllie suggests to ask the patient first to write his name, and, if he succeeds, to put simple questions to him and ask him to answer them in writing; if he writes with comparative ease, to ask him to write at his leisure the history of his illness, and to note in his performance evidence of—(1) paragraphia (which is usually only the written translation of paraphasia); (2) intoxication of the mind with a letter or a word; and (3) faults of spelling or of syntax. If the right hand is paralyzed, he suggests to let the patient try to write with the left; if the right

hand is not paralyzed, to let him furnish examples of his performance in writing with both the right hand and the left. He should be tested not only as to whether he understands printed or written letters, but also as to his comprehension of such graphic symbols as numbers, algebraic signs, and musical notes. Of course an examination in these directions would be necessary or not according to the previous education of the patient. He should be studied as to his powers of comprehending verbs and nouns, as well as to his methods of using these parts of speech. In examining for verbal amnesia, the patient should be shown common objects and asked to name them. If he is completely speechless, it should be learned whether or not he is able to write the name of the object which is shown him. If he can neither speak nor write, he may be tested as to his ability to express his meaning through pantomime. Following the method of Lichtheim, for instance, he can be asked to indicate with his fingers the number of syllables in the name of the object shown to him. He can be tested also as to psychical deafness by observing whether or not he recognizes the use of objects about which he is told. In studying motor aphasia, if a patient can speak only a few words or expressions, what these are should be noted, and whether his utterances are of a recurring character; also whether he uses words with their proper meaning, as "yes" for "yes" and "no" for "no." If he is able to talk, the character of his utterance should be observed, and the manner in which he uses his organs of articulation; it should also be observed whether he has any paresis of the face or tongue or of the facial muscles. He should be tested not only as to whether he understands speech, but also as to whether he understands music and musical tones. It should be observed whether the patient repeats or echoes the words spoken to him, and also, if he does this, whether he seems to understand the words which he repeats or echoes. He should be thoroughly examined for pantomime, after the methods indicated in the brief discussion of this subject. The investigator should constantly keep before his mind that he is to determine not only what the patient has lost, but also what he retains, of language or of thought. His general mental condition should be carefully considered. The physician should satisfy himself that the patient is not suffering from stuporous melancholia, nor from dementia, nor from hysterical mutism, nor from any of the forms of nervous or mental diseases which may impair his language or render him mute.

Diagnosis.—*Differentiation of the Clinical Varieties of Aphasia.* Much that has been said in describing the different forms of aphasia will be of value in separating the varieties of speech defects from one another,—the sensory from the motor, both of these from the conceptual forms, and the different varieties of sensory and motor aphasia from one another. Word deafness is readily separated from

motor aphasia, but the one may be complicated with the other when large lesions are present. If it is clear that noises or sounds are heard, but that the words cannot be perceived as such, it is evident that verbal deafness is present. The patient does not respond to verbal stimulation. Complete cerebral deafness may result from double or successive lesions in the superior portions of the temporal lobes, and it might be difficult to tell whether the patient was or had been word deaf. The history of the case would be of most value. The existence of peripheral deafness is, of course, determined by the ordinary methods of aural examination. Verbal deafness may be complicated with both motor aphasia and verbal blindness, when it may be difficult to determine whether the patient is suffering from true verbal deafness, or is deaf, mute, or demented. Such a complication is rare, and would most likely be due to multiple lesions. The confusion and irritability of the patient would add to the difficulty, and the problem could be solved only by a close analysis of the symptoms and the method of their appearance. The history of apoplectic attacks, the association of paralysis with speech disturbances, and the existence of previous mental health would be against the case being one of melancholia or dementia with deafness and mutism.

Differentiation of Cortical from Subcortical and Subcerebral Aphasia. A study of the patient's power of pantomime will sometimes assist in the diagnosis of cortical from subcortical aphasia. When, for instance, a patient totally aphasic so far as articulate words are concerned is asked how old he is, or how many syllables are in a word, if his cortical centres for motor images are intact he may be able to answer in pantomime, by opening and closing his hand a certain number of times, or by lifting the hand and letting it fall, or by a certain number of nods of the head. Speech disturbances are sometimes present and marked in lesions of the pons, and especially when these lesions are bilateral. These disturbances are similar to those which have been described as present in cases of subcortical motor aphasia. They are of the nature of anarthrias. The diagnosis from subcortical aphasia due to cerebral lesion would be made by a consideration of the other symptoms and signs of pontile lesion.

Differentiation of Different Forms of Agraphia. It is hardly necessary to say that in the diagnosis of agraphia the patient's education and previous ability to write must not be overlooked. It may, of course, be difficult to tell in hemiplegic aphasics whether or not the agraphia is dependent upon the paralysis and contracture. The true agraphic will not at first be able to write either with the unparalyzed or with the paralyzed hand, and if he tries with the unaffected member he may write in mirror fashion. If he is a true motor agraphic, even though he retain some power in his partially paralyzed hand and fingers, he cannot write, or even in most cases make an effort to

write, with the paralyzed hand. A cortical motor aphasic if he preserves the power of copying with either hand usually copies the words very exactly, but turns the printed letters into script; the sensory aphasic copies mechanically with great difficulty letter by letter, as one would reproduce any figure or picture before him. Motor agraphia can be differentiated from the agraphia which is due to lesion of the visual centres by the presence of other symptoms which indicate involvement of the sensory or receptive side of the brain, such as word blindness and letter blindness. Sérieux has reported a case of word blindness with agraphia in which the left inferior parietal lobule and angular gyre were softened; the patient had no paralysis, no hemianopsia, and her general vision was good; she could scarcely make out a single letter. Although the movements of the right hand were perfect, she could not write a word legibly. Writing from dictation and from copy, as well as spontaneous writing, was very difficult.

Prognosis.—The prognosis of a case of aphasia varies according to the severity of the attack, the pathological nature of the lesion causing it, and, to some extent, the variety of the aphasia. Attacks due to large and destructive lesions of the cerebral substance are, of course, always of comparatively unfavorable omen. Aphasia, which may be complete and is often of the motor or mixed form, sometimes persists for years or until death, the patient either recovering no speech or at at best recovering only a few stock or recurring expressions. Such cases are usually associated with a large amount of paralysis. It is not wise, however, to decide too early that a case of complete hemiplegia with nearly complete aphasia will have an entirely unfavorable prognosis. The aphasia in these cases is sometimes due to compression or the effects of cerebral shock, and when this passes away speech is rapidly recovered. The existence of a large destructive lesion permanently affecting the speech centres and tracts can be determined with some approach to accuracy after the acute and subacute symptoms of the apoplectic attack have subsided. In cases which are due to localized syphilitic lesions, as to a gumma, to localized meningitis, or to encephalitis, the prognosis is comparatively good unless the specific lesion has persisted sufficiently long and its invasions have been such as to cause considerable destruction of brain substance. On the whole, the prognosis of a case of aphasia due to hemorrhage is somewhat better than when it is due to acute softening from embolism or thrombosis. As to the variety of aphasia, much that has already been said indicates that some of the forms have a more hopeful outlook than others. The receptive and interceptive or conceptual aphasias are, on the whole, more likely to improve, at least to make partial and in some cases considerable improvement, than are those of a motor or a productive character. Word blindness, often at first complete or nearly complete, may in a

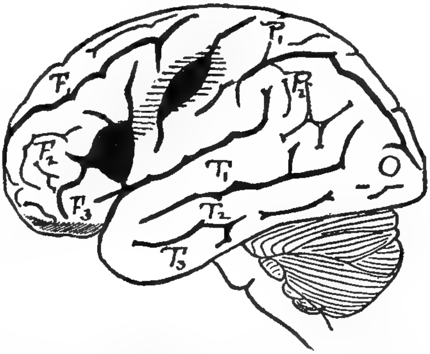
large degree pass away, while the paraphasia and the motor aphasia, if the case be of mixed type, largely persist. Complete word blindness, unless transient and partial, is not often recovered from entirely. Much, of course, will depend on the extent of the loss of language and on the previous intelligence of the patient. Thomas and Roux, after a study of seventeen cases of cortical motor aphasia, concluded that the patient in the course of improvement first recognizes the appearance of the word, then associates the syllables which form the word, next the letters which form each syllable of each word, and at last the meaning of the whole word. In other words, such an aphasic patient recovers the ability to read in exactly the inverse order in which a child learns to read. Aphasia of the most complete type and of almost any variety is sometimes due to organic but evanescent disease of the brain or its membranes, as to meningitis, encephalitis, or meningoencephalitis; or it may be dependent upon concussion, compression, or contusion of the brain. In compression and concussion, extravasations of blood are sometimes present either in the interior of the brain or above or below the dura. Inflammation subsides, or absorption of the extravasation takes place, and the patient recovers in whole or in large part from his speech as from other disturbances. Whether aphasia is due to hyperemia of the brain is regarded as doubtful by some. Many cases of evanescent aphasia were formerly attributed to congestion of the brain; but often the cases so explained were due to other causes.

Treatment.—*Medical Treatment.* The patient may be treated either medically or surgically for the affection which has caused the aphasia. If he has a syphilitic history, the possibility of a tumor should be considered, and mercury and the iodides should be used as in other cases of cerebral syphilis. Even when the disease attacks chiefly the vessels, such treatment may be of some avail in preventing its rapid inroads. The nutrition of aphasic patients should receive careful attention, as the weakened and injured brain needs to be nourished and supported. Occasionally aphasia may be due to gouty inflammation of the membranes, or, as has been shown in a previous paragraph, to localized or diffuse hyperemia. In such cases the medicinal treatment should be in accordance with the condition supposed to be present. Lithium and colchicum preparations can be given in the gouty cases, and local and general depletion used with the bromides, hydrobomic acid, ergot, and drugs of a similar kind, to relieve the congestive attacks.

Surgical Treatment. Some of the most interesting and successful cases in the history of cerebral surgery have been those in which aphasia has been relieved as the result of trephining. Surgeons should be able to recognize the types and subtypes of aphasia. The centres and tracts for speech are so far apart that the trephine needs to be placed in distinctly different regions, separated in some cases

by several inches. In some head injuries with aphasia, in which the nature of the aphasia, and therefore the site of the lesion causing it, can with a fair degree of certainty be fixed, it may nevertheless be best to wait a few days before operating. Such a case may be due to contusion, hemorrhage, or fracture, and in a short time the aphasia may pass away. In a case seen by me in consultation with Dr. Deaver at the German Hospital, the patient had marked motor aphasia, paraphasia, paralexia, and paragraphia, associated with paresis of the lower portion of the right side of the face, and evidences of injury in other parts of the body. Operation was postponed, although the lesion was regarded as probably a meningeal or cortical hemorrhage implicating Broca's convolution and the face centre. In about a week the aphasia began to disappear, and soon the patient entirely recovered. Other recorded cases, however, have shown the importance of not postponing operation. (Fig. 339.) Macewen, for instance, reports a case of hemiplegia, aphasia, and convulsions in which trephining was proposed, but, after a consultation, was negatived. The patient died, and after his death trephining was performed in the same position as had been suggested during life, namely, over the left subfrontal convolution, and an abscess was revealed which occupied chiefly the white matter of the posterior extremities of both the second and third frontal convolutions. Aphasia and forms of cerebral paralysis occasionally occur after operations. The records show, for instance, the occurrence of such cases after amputations, after the removal of hemorrhoids, and after operations about the face. Allied to these cases are those of paralysis and aphasia which now and then are observed after labor or during the lying-in period.

FIG. 339.



The left hemisphere of the brain, showing situation of clots causing motor aphasia and right hemiplegia in a case successfully operated upon. (Starr.)

Treatment by Training. The most important matter in the treatment of aphasia is that of training, which must differ according to the variety. Many cases improve greatly without training, but the importance of persistent efforts in this direction has been strongly impressed upon me by the results of my observations in several cases. A few years ago I carefully studied a case of transcortical and motor aphasia of very complete type. The patient was a determined man of considerable intelligence, and made persevering and successful efforts to reeducate himself in both speaking and writing, being

spurred on by his own ambition and the interest manifested by others. He improved so that he could talk, read, and write legibly and intelligently, although with some awkwardness at times in his mode of expression. A few special words need to be said about the methods of training in the different varieties of aphasia, a subject to which Wyllie has given considerable attention. In the treatment of auditory aphasia words should be slowly and clearly pronounced in the hearing of the patient, who should be asked to repeat them, and he should also be asked to read printed words. It is probable that in this way the word images are sufficiently imprinted in the uneducated auditory centres and at the same time connections of the requisite intimacy are established between these images and those of the other speech centres. (Wyllie.) Often verbal blindness is susceptible of improvement, although when due to destructive lesion it is probably never entirely cured. In a few cases the preservation of the auditory and motor memories enables the visual memory to be partly re-educated. In the attempts to treat patients suffering from literal or verbal blindness, printed and written letters and words should be shown to them, and they should be told what they are and asked to repeat them. The same letters or words should be shown at other times and in other relations, until the patient learns to recognize them through the formation of the visual images. When patients suffer from verbal amnesia and from apraxia, objects should be shown and named, the patient being asked to repeat the names. In some of these cases of verbal or psychic amnesia without verbal blindness or deafness or motor aphasia, considerable and sometimes rapid improvement is made; in other cases the improvement is slight. Efforts at training a motor aphasic, if conducted with great patience, often give favorable results. At first the patient should be made to repeat letters and simple words. Step by step more difficult words and simple phrases and sentences should be tried. Certain principles can be followed with advantage in this process of training. Wyllie suggests that if the patient can be made to master the letter sounds it will be easier for him afterwards to produce their combinations in the form of words. In one of his cases he adopted the "mother's method." Beginning with the labials, the aphasic was first taught to say *papa*, *apap*, *appa*; in this way the consonant *p* was used as an initial, a middle, and a terminal letter. Next he was taught to say *baba*, *abab*, *abba*; then *mama*, *amam*, *amma*; then *weewee*; and so on throughout the alphabet. He was then shown by lip reading how to place the lips and tongue in producing different letter sounds. Next he was supplied with children's books of the primary sort, from which he learned to read aloud short sentences. The progress of this patient was greatly accelerated by this method, which is one to be recommended for other motor aphasics, and has been used by the author.

CHAPTER VII.

AFFECTIONS OF THE SPECIAL SENSES DUE TO LESIONS AND DISTURBANCES OF THE NERVES OF SPECIAL SENSE AND OF THEIR CORRELATED CENTRAL STRUCTURES.

ENUMERATION OF THE CRANIAL NERVES.

At the present day the number of cranial nerves is almost universally given as twelve pairs, as follows: first, olfactory; second, optic; third, oculomotor; fourth, pathetic; fifth, trigeminal; sixth, abducent; seventh, facial; eighth, auditory, including both cochlear and vestibular; ninth, glossopharyngeal, including the pars intermedia of Wrisberg; tenth, pneumogastric; eleventh, spinal accessory; and twelfth, hypoglossal. At least two or three of the cranial nerves seem to demand subdivision. With the exception of the olfactory and the optic, all the cranial nerves arise from either the midbrain or the hindbrain, and their characteristics are largely those of the spinal nerves. The plan pursued in the chapters which immediately follow differs considerably from that of most neurological textbooks. In the first of these chapters will be considered the affections of special sense dependent upon lesions and disturbances of the nerves of special sense and of their correlated central structures.

AFFECTIONS OF SMELL DUE TO DISEASE OF THE OLFACTORY NERVE AND OF ITS RELATED ENCEPHALIC STRUCTURES.

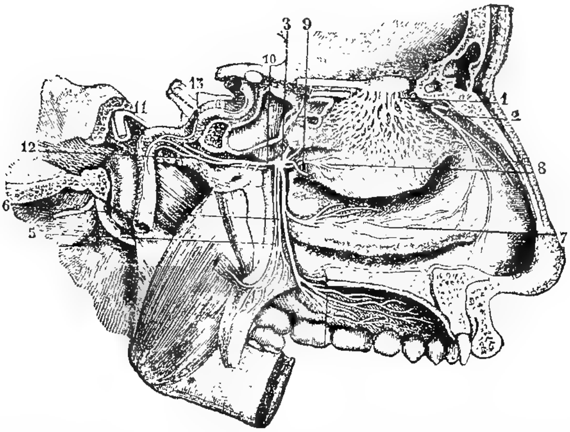
The Olfactory Apparatus.—*Summary of the Olfactory Paths and Centres.* The anatomy and functions of the olfactory nerve and the location of the cerebral organ of smell have been partly described in previous sections (see the comparative table showing the constitution of the different sensory tracts and their homologues, page 105, and the discussion of olfactory localization, pp. 347–348). It has been shown that the olfactory tract begins peripherally in the bipolar cells of the mucous membrane of the nose, passes to the mitral cells in the olfactory bulb, and thence to the precommissure and to the olfactory roots and fornix. The short or reflex central path of the nerve, if such exists as a distinct path, is probably by way of the albicans and anterior portion of the thalamus; while the cortical regions or centres which receive the olfactory fibres are, as has been indicated, the precallosal part of the gyrus fornicatus, the septum lucidum, and the inferior extremity of the hippocampal gyre and the uncinate gyre, and probably also the amygdala, the dentate fascia, and the striæ

of Lancisi, which terminate in the dentate fascia. That the cortical centres for the sense of smell are in large part situated in the temporal lobe is conclusively shown by the simplest studies in the comparative anatomy of osmatic and anosmatic animals. In osmatics, such as the dog, wolf, jackal, and other animals which follow their prey with the nose, the temporal lobe is larger and more projecting than in animals which depend mainly upon their hearing, like the tiger, panther, leopard, and the other felines, and those which rely chiefly on the sense of sight, like the herbivorous animals. The largest portion of the reptilian central cortex is olfactory. (Edinger.) The brain of the roothunting pig takes an intermediate position, as far as the development of its temporal lobe is concerned, between the true osmatics and the anosmatics. The Sylvian fissure in these animals differs in its verticality and in the manner in which its lobes separate in accordance with the relative development of the temporal lobe. The olfactory apparatus in man is in a comparatively atrophic condition, a fact which cannot be too closely kept in mind when studying the anatomical structures and extensions of the olfactory centres and tracts. As many of the observations on the anatomy and physiology of the olfactory apparatus are based upon studies of the lower, and especially of the osmatic, animals, the deductions to be drawn from these investigations must be made with great care. It is now generally believed that the olfactory and optic nerves have a significance different from that of the other cranial nerves,—that they are best regarded as portions of the central nervous system. In many lower animals the olfactory bulb appears as a large lobe attached to the base of the forebrain. While, however, the olfactory and optic nerves partake less of the character of ordinary peripheral nerves than do the other cranial nerves, and while they are in a certain sense to be regarded as integral parts of the brain rather than as true peripheral nerves, they are structures which put certain parts of the cerebrum in relation with the outside world, in this way performing functions which are performed by other cranial nerves. Both the olfactory and the optic nerves arise from the anterior primary vesicle of the forebrain,—the olfactory bulbs and tracts, however, originating from the first secondary vesicle, or prosencephalon, the optic from the second secondary vesicle, or diencephalon.

Peripheral Portion of the Olfactory Apparatus. The olfactory nerve arises by a score or more of slender filaments which pass from the mucous membrane of the nose to the olfactory bulb. Their course is through the numerous foramina in the cribriform plate of the ethmoid bone (Fig. 340). By some these filaments are divided into inner and outer groups. The origin of the olfactory nerve is of the simplest character, resembling more closely than any of the other nerves of sense that of the earthworm. The olfactory filaments arise in the mucous membrane covering the superior turbinated bone, the upper

third of the middle turbinated bone, and the upper part of the septum. These filaments pass through the mucous membrane from their origin in bulbous expansions called olfactory cells. The olfactory epithelium represents far more the origin of the olfactory fibres than it does their termination. Recent investigations have shown that in addition to the fibres that arise in the olfactory cells free intraepithelial nerve terminations occur, so that the regio olfactoria, like the retina, sends fibres towards the brain and receives fibres from it.

FIG. 340.



View of the sphenopalatine ganglia, the outer wall of the left nasal cavity, and the olfactory nerve. 1, external olfactory nerves; 2, nasal branch of the ophthalmic nerve; 3, sphenopalatine ganglion; 4, 5, 6, palatine nerves; 7, inferior nasal nerve; 8, superior nasal nerves; 9, nasopalatine nerve; 10, Vidian nerve; 11, facial nerve; 12, deep petrosal nerve joining the carotid plexus, 13; the other branch of the Vidian is the superficial petrosal nerve, which joins the facial. (Leidy, after Hirschfeld and Sappey.)

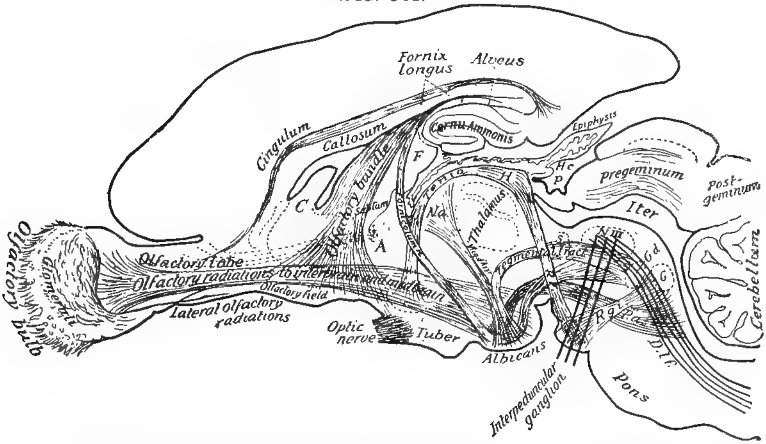
Brunn considers these terminations as belonging to the trigeminus, and states that they are thicker than the olfactory fibres. Lenhossék has also observed these fibres in the fetuses of rabbits. They appear as unbranched, delicate, and smooth fibres which ascend perpendicularly between the olfactory cells and the surface of the epithelium, terminating in small enlargements. They resemble the olfactory fibres; and Lenhossék doubts the view of Brunn that they are trigeminal fibres. Ramón y Cajal later found these fibres in the fetuses of mice in Jacobson's organ, as well as in the olfactory mucous membrane. Lenhossék in 1894 described free terminal fibres within the olfactory mucous membrane of a mouse of eight days; but this type he refers to the trigeminal fibres. This investigator believes that there are probably two kinds of fibres under and in the olfactory mucous membrane, one undivided, probably olfactory, and the other divided, and probably belonging to the trigeminus. Kallius found them in the regio respiratoria and considers that they are trigeminus fibres. In a strict sense only the filaments which pass through the

cribriform plate of the ethmoid bone constitute the olfactory nerve. The olfactory tract from the bulb to its cortical centres is morphologically and anatomically analogous to the optic tract. The olfactory filaments (*nervuli olfactorii*) are the peripheral neurons or nerve cells which correspond to the nerve cells of the intervertebral ganglia. They pass to the olfactory glomerules of the olfactory bulb (*glomeruli olfactorii*), and in these the peripheral portion of the olfactory path may be regarded as ending. The olfactory bulb, as described by Edinger, shows in section most externally a gray ganglion cell layer which gradually merges through a granular zone into the olfactory marrow or white substance. As shown by embryological studies, the fibres of the olfactory nerves are nothing but the centrally directed terminal fibres of the sensory cells of the olfactory mucous membrane. These terminal fibres, after passing through the cribriform plate of the ethmoid bone, continue along the ventral surface of the olfactory bulb, where they cross and recross, and eventually pass onward into the substance of the olfactory bulb. In the brain substance each axis cylinder breaks up into fine terminal ramifications. The olfactory glomerules are made by the juxtaposition of these ramifying fibres with other similar ramifications, of more central origin,—dendritic processes which arise from ganglion cells. Each of these ganglion cells of the bulb is in communication with several nerve fibres. These ganglion cells have also nerve processes or axis cylinders which may be followed into the brain, and which send out occasional collaterals on their way. In lower animals the olfactory bulb is placed directly upon the so-called olfactory lobe, and caudad spreads outward into the quadrilateral space of Broca.

Central Portion of the Olfactory Apparatus. Nerve tracts pass from the olfactory bulb to the olfactory lobe and to other structures entering into the composition of the olfactory apparatus. In the glomeruli begins the central portion of the olfactory path (the central neurons). The precommissure, the septum, the fornix, the albicantia, the bundle of Vicq d'Azyr, the habenula, and other tracts and centres, the course of which will now be briefly described, constitute portions of the olfactory apparatus, or rhinencephalon. An understanding of this description will be assisted by a study of Fig. 341, in which is shown diagrammatically from Edinger with modifications from Koelliker a sagittal section of the brain of a rabbit, made a little to the left of the mesal line. Fibres from the olfactory bulb and lobe are seen passing to the precommissure, the septum, the horn of Ammon, the habenula, and the albicans. From the albicans several bundles are seen to pass, as the anterior column of the fornix, the tract of Vicq d'Azyr, a bundle to the tegmentum, and one to the pons. The precommissure (anterior commissure) is certainly the commissure of the rhinencephalon, and has its greatest development in the macrosmatic mammalia, while it is poorly developed in the

microsmatic, as in man, and in the anosmatic, as the dolphin, it is scarcely present. It has been positively determined by Koelliker that a considerable portion of the fibres of the precommissure terminate with extensive ramifications about the mitral cells and in the molecular zone of the olfactory bulb. He also succeeded in demonstrating free terminations of precommissural fibres in the pyriform lobe. According to Koelliker, the portion of the commissure in connection with the lobus pyriformis arises in the pyramidal cells of one lobe and ends on the other side with free terminations about such cells

FIG. 341.



A sagittal section of the brain of a rabbit, a little to the left of the mesal line. A large number of the fibres drawn fall into the plane of the section and have been drawn from the preparation made in this plane. A smaller portion, including the fornicolumn and others, have been drawn as if in the same plane, but in reality from sections made a little more laterally. *C*, head of caudatum; *A*, precommissure; *F*, fornix and psalterium; *N.a.*, anterior nucleus of the thalamus; *R.f.*, fasciculus retroflexus; *N.III*, nucleus of the third nerve; *P.a.*, peduncle of the albicans (pedunculis mammillaris); *D.l.f.*, dorsal longitudinal bundle; *H*, ganglion habenulæ; *H.c.*, commissure of the habenula; *P*, postcommissure. (After Edinger and Koelliker.)

or their dendritic processes, and *vice versa*. In the olfactory bulb apparently only those cells can be considered in this connection which form the different varieties of brush cells (*pinselzellen* of the German writers). It is certain that the large brush cells send their axis cylinders into the tractus olfactorius, while such a condition cannot be assumed with certainty for the smallest cells which surround the glomeruli. A great tract of fibres passes backward from the cortex of the olfactory field to the septum (*septum lucidum*). In the septum some of the fibres decussate and others go directly backward; the decussating and nondecussating bundles unite and pass into the fornix from the caudal border of the septum. Some of the fibres continue backward in the fornix into the white matter of the horn of Ammon, and others through the callosum into the gyrus cinguli. These may be regarded as centripetal. The fibres from the fornix and others, as the olfactory bundle, commingle in the fimbria. A

portion of the olfactory fibres pass forward from the fimbria and descend into the fornicolumn, to the albicans. A long tract of thick fibres, described by Forel as the *fornix longus*,* rises out of the gyrus fornicatus and the superficial layer of the horn of Ammon and from the subiculum, and, lying close to the callosum, turns downward and divides into two bundles, of which the posterior and chief passes into the anterior pillar of the fornix, the other division into the septum. Koelliker believes he has found the fornix longus in man. The lyra or psalterium, sometimes designated as Ammon's commissure, is probably a portion of the olfactory apparatus. The fornicolumn makes a bend or knee where it enters the albicans. Some of its fibre tracts pass through and beyond the albicans; others end among its cells. The tract from the albicans to the thalamus, known as the bundle of Vicq d'Azyr, or fasciculus thalamomammillaris (Koelliker), is a portion of a bundle or tract which originates in the albicans. After leaving the dorsal side of the albicans this bundle almost immediately divides at right angles, one part becoming the bundle of Vicq d'Azyr and the other the tegmental tract. Koelliker found that in two series of frontal sections from man he could trace into the dorsal longitudinal bundle fibres of the tegmental tract, or at least he could not distinguish the fibres of this tract from those of the fasciculus longitudinalis dorsalis. According to Koelliker, the habenula is undoubtedly a portion of the apparatus for smell. It is, in the first place, in connection with the olfactory tract by means of fibres which enter it from the fornix, also probably by means of a portion of the striæ medullares,† which run from the basal ganglion near the

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* The diagram on page 671 and the description of the olfactory path are based largely upon the work of Forel, Van Gehuchten, Edinger, and Koelliker. Recently, G. Elliot Smith (Journal of Anatomy and Physiology, Normal and Pathological, vol. xxxi. (N. S., vol. xi.) Part 1, October, 1896) has challenged some of these views in a contribution on the *Fornix Superior*. He failed to establish the origin of the fornix longus from the gyrus fornicatus in man, and in regard to the lower animals, and especially the marsupials, he believes that it can be definitively said that no such connection exists. He admits that the fornix being the associating system of the hippocampus must be partly olfactory in function, but as it exists in a well developed form in anosmatic animals it cannot be wholly olfactory. He believes that the olfactory bundle of Zuckerkandl (see diagram Fig. 341) is mainly nonolfactory, since it exists in a well developed form in anosmatic animals. He reasons that, since the fornix is a heterogeneous system of fibres, one is not justified in concluding that a region of the cortex is olfactory in function because it is connected with the fornix. Smith claims to have discovered the fornix longus in man before Koelliker's observations were published.

† "Striæ medullares" is one of the anatomical terms used in designating different and unrelated structures, as in this case, and when employed as synonymous with the striæ acusticæ which traverse the floor of the fourth ventricle. It would be better not to use the same designation for different anatomical structures or organs, but the term is retained here because it is in general use among neurologists.

chiasm. That the ganglion habenulæ is connected with the optic tract is shown, according to Koelliker, by facts of comparative anatomy which speak for the relation of the pineal gland to the parietal eye of the lower vertebrates. These connecting tracts Koelliker refers in the first place to fibres of the stratum zonale, which end in the mesal nucleus of the ganglion habenulæ. Mendel and Darkschewitsch have asserted the existence of this connection between the ganglion habenulæ and the optic tract. Koelliker's general hypothesis is that the olfactory neurons of the second order send their axis cylinders through the olfactory radiations of the septum, through the fornix longus of Forel around the knee of the callosum, through the callosum into the gyrus cinguli, and also through a second portion of the fornix into the horn of Ammon. Between the granule cells of the fascia dentata are also terminations of the olfactory neurons. The diagram Fig. 342 is intended to represent the falciform or limbic

FIG. 342.

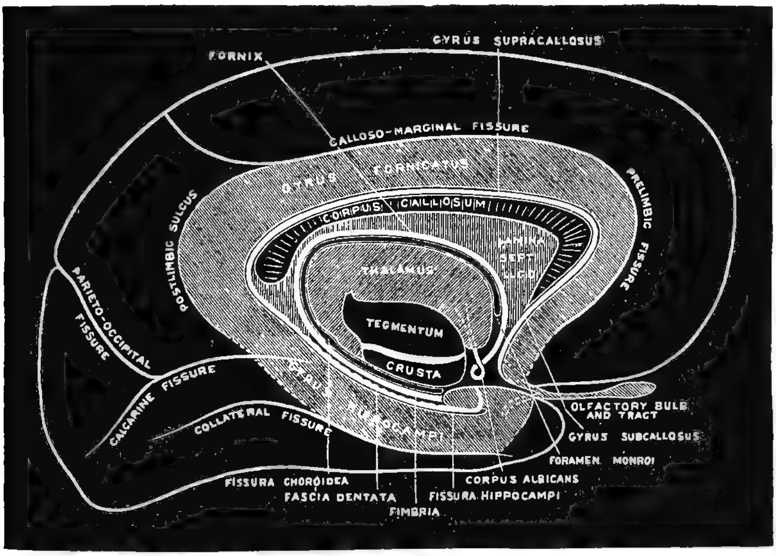


Diagram of the falciform lobe of Schwalbe (limbic lobe of Schafer). This diagram indicates most of the cerebral structures the whole or part of which constitutes the olfactory apparatus according to the researches of Edinger, Ramón y Cajal, and Koelliker. The parts not shown referred to in the text are the striæ Lancisi and some of the centres in the interbrain and midbrain. These and the fibre tracts are indicated in the previous diagram from Edinger. (Schäfer, in Quain's Anatomy.)

lobe, and illustrates largely the parts described in the preceding discussion of the olfactory apparatus. It must be remembered that the whole of the gyrus fornicatus is not regarded in man as concerned with olfaction alone. The precommissure, the striæ Lancisi, and the habenula are not indicated. Fibre tracts are shown in the preceding figure.

Historical.—In A.D. 800 a learned monk with the sesquipedalian name of Protospatharius wrote a work on the parts of the human body which was equal in anatomical exactness to the usual works of that period, but in it he first correctly indicated that the first cranial nerve was concerned with the function of smell. Galen believed that odors passed bodily into the cerebral substance by means of the olfactory nerve, which he regarded as a canal. Vesalius was the first to dissect the olfactory nerve. Lobestein first observed the third central gray root. The great physiologist Magendie held that the fifth and not the first nerve was the principal organ of smell, his opinion being founded chiefly, if not entirely, upon experiments upon animals. The substances used to test the sense of smell were probably of a pungent and volatile character. Claude Bernard taught that the question was unsettled, but abundant evidence has been adduced in recent years to prove conclusively that the first cranial nerve is the true organ of smell. (Althaus.*) Cases have been reported in which the sensibility of the nasal mucous membrane has been completely blunted and yet the patient has retained perfectly the sense of smell. Numerous physiological experiments have conclusively shown the relations of the olfactory nerve to smell; and histology and comparative anatomy have all added strength to the same view, which indeed it is more interesting than valuable to discuss.

Clinical History.—*Varieties of Olfactory Disease.* While much attention has been paid to the study of the comparative anatomy and physiology of smell, clinical and pathological data are not abundant. Olfactory affections may be discussed as (1) focal lesions affecting the olfactory path, such as tumors, hemorrhage, acute softening, encephalitis, abscess, aneurism, basal meningitis, caries, or exostoses; (2) olfactory neuritis; (3) congenital absence of the bulb, or arhinencephalia; (4) primary degeneration of the olfactory nerves and tracts; (5) hysterical or other functional affections of smell; and (6) nasal olfactory neuroses. Affections of smell may be unilateral or bilateral, and may be partial or complete on either one side or both. Destructive lesions cause loss of smell, and irritative lesions perversions of smell; but sometimes loss or impairment of smell may be due to pressure affecting the olfactory tracts or centres.

Olfactory Terminology. Loss of smell is known technically as *anosmia*, its synonyms being *anosphresia*, *anosphresis*, or *anosphrasia*. Hyperacuteness of smell is designated *hyperosmia*, *hyperosphresia*, or *hyperosphresis*. When the sense of smell is perverted, the disorder is known as *parosmia*, *parosphresia*, or *parosphresis*. *Cacosmia* or *kakosmia* is an affection in which the patient experiences disagreeable or disgusting odors. For the illusions and hallucinations of smell either

* These historical and other facts have been obtained from an interesting paper by Dr. Julius Althaus in the *Lancet* of May 14, 1881.

the term parosmia or the term hyperosmia is sometimes used, but the former is preferable, as its proper interpretation is perversion of smell rather than simple increase in acuteness.

Special Olfactory Symptoms. In tumors and other irritative lesions affecting the olfactory nerve tract or centres, the symptoms are first those of hyperosmia and parosmia, and later anosmia, partial or complete. Similarly, in olfactory neuritis at first hyperacuity of smell and hallucinations of odors may be present, and subsequently loss of smell. A few remarks may be made as to the symptoms in any case of anosmia. The sense of smell may be lost either suddenly or slowly, according to the nature of the causative lesion. The sense of smell differs normally in different individuals. Often, particularly in unilateral and partial cases, the affection is not recognized by the patient until his attention is called to it by some special circumstance. If the loss is abrupt and complete he is more apt to recognize it at once. Among those who follow occupations in which they are constantly exposed to offensive odors, a form of tolerance to smells is induced which becomes practically a partial anosmia. Occasionally a true anosmia results in these cases from the continued excessive stimulation of the olfactory organ. Forms of rhinorrhea of nervous origin are sometimes associated with parosmia.

Flavors and Odors. Flavor is dependent upon the joint action of the senses of smell and taste; hence one who has lost the sense of smell may think that he has lost also that of taste, because he is unable to distinguish the flavor of the food or drink of which he partakes, although he can distinguish such substances as salt, sugar, quinine, and citric acid. Odors alone are lost through disease of the anterior nares, both odors and flavors when the disease affects the posterior nares. When loss of smell is suspected or complained of, the patient should be carefully tested both for this sense and for that of taste. The methods of testing for smell and taste have already been given (pages 164–165). Care should be taken to discriminate between affections of smell and those of general sensibility the result of disease of the nasal distribution of the fifth nerve. Even when the sense of smell is lost, a volatile, pungent substance, like ammonia, may be distinguished through the trigeminal nerve and deceive the careless investigator. Froelich distinguishes odors acting purely on the olfactory nerve, such as essential oils, resins, and balsams, from those acting likewise on the fifth nerve, such as iodine, bromine, chlorine, nitric acid, strong acetic acid, ammonia, horseradish, and mustard. The former act simply as odors, while the latter irritate the mucous membranes of the eyes, nose, and throat, and cause lachrymation, sneezing, and coughing.

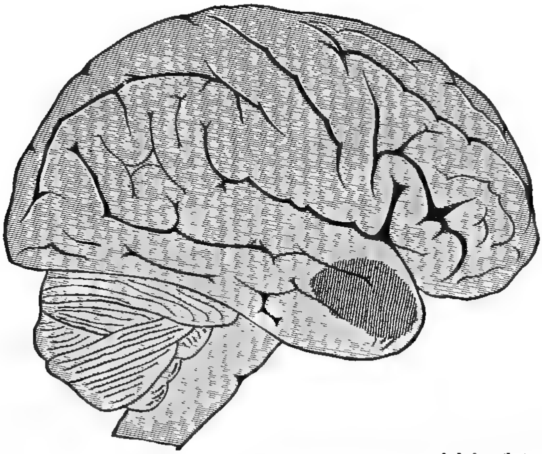
Hallucinations of Smell. Curious forms of parosmia are met with even among the sane, as in the case of a man to whom violets always seemed to smell like phosphorus, and another to whom mignonette

had the odor of garlic. Hallucinations of smell are comparatively common among the insane. Most frequently they occur in connection with other hallucinations, as of taste or touch ; sometimes they are the results of suggestion. In general paralysis of the insane and in other organic insanities they may be dependent upon lesions of the cortical centres or tracts for smell. Loss of appreciation both of pungent substances and of true odors is sometimes present in general paralysis. Hallucinations of smell may sometimes constitute a portion of the initial state of a case of melancholia, mania, or paranoia, and, as is well known, they may take the form of aura in epileptic insanity or may be present during the interval between the epileptic paroxysms. In rare instances the olfactory sense is excited during dreams ; but neither this sense nor that of taste is, as a rule, aroused, even when the dreams refer to occasions in which taste and odors have played an important part, as when they are of banquets and convivial occasions.

Focal Lesions affecting the Olfactory Apparatus. Tumors of the prefrontal lobes and of the anterior fossæ of the skull frequently cause impairment or loss of smell, hyperosmia or parosmia sometimes occurring in the progress of the case. Anosmia may be present when the olfactory bulb and the olfactory filaments are only indirectly implicated in the lesion, as when growths in the centrum ovale or in the lateral or convex portions of the prefrontal lobe affect the olfactory bulb by pressure. If careful investigation is made, the affection of smell may be found in rare cases to be unilateral, although a large growth on one side frequently causes involvement of both olfactory nerves. A considerable number of cases of gross lesion involving the prefrontal lobe and giving disorders of smell have been placed on record. In these cases, when the olfactory disorder is not due to implication of the olfactory filaments or bulb, the extension of the lesion may be such as to involve some of the central portions of the olfactory path. Tumor cases have been most frequently recorded, but in other instances hemorrhage, acute softening, encephalitis with or without abscess, aneurism of the internal carotid, precerebral, or medicerebral artery, basal meningitis, or bone disease, has been the exciting lesion. From what has already been said about the olfactory path, it is evident that a focal lesion situated in the ventricles or ganglia, or so extending from any situation as to involve the fornix, albicantia, thalamus, or any of the basal olfactory tracts or centres, would give rise to disorders of smell. When discussing lesions of the thalamus (page 355) a case was referred to in which smell, sight, hearing, and general sensibility were successively lost as the result of a neoplasm which gradually destroyed both thalami. A critical study of the sense of smell is not infrequently overlooked in cases of intracranial focal lesion which involves the ganglia, ventricles, and central regions of the

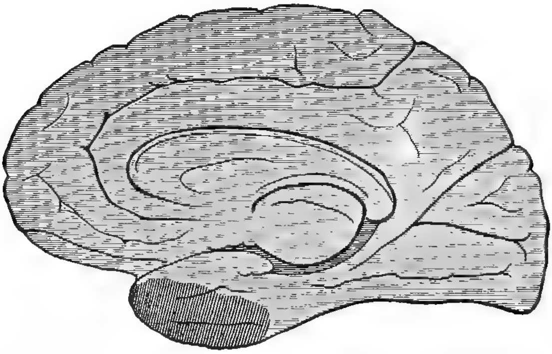
base, but it is probable that anosmia is often present. A few cases have been reported in which anosmia and various perversions of smell were present as the result of lesions in some of the cortical regions which have been described as the cerebral centres for smell. Cases of tumor and of softening involving the anterior mesal portions of the temporal lobe with aura and hallucinations of smell,

FIG. 343.



Glioma of the anterior and inferior portions of the right temporal lobe (lateral surface) with parosmia as a prominent symptom. (Coupland.)

FIG. 344.



Glioma of the anterior and inferior portions of the right temporal lobe (mesal view) with parosmia as a prominent symptom. (Coupland.)

and in some instances with anosmia, have been reported. It is not infrequent to have epileptic paroxysms initiated by an odor commonly of an offensive character, and in some of these cases this symptom may be due either to macroscopic or minute lesion of the cortical areas for smell. Coupland has described a case in which autopsy showed gliomatous infiltration of the anterior and inferior portion of the right temporal lobe. This patient had suffered from severe

epileptiform attacks. Following the first of these, which was slight, he complained of a very offensive smell, and at intervals after this of the same olfactory derangement. The position and extent of the growth in this case are shown in Figs. 343 and 344.

Olfactory Neuritis. It is almost impossible to demonstrate the existence of an inflammatory affection of the olfactory nerve filaments or their terminations, or of the intracranial olfactory tract; but a condition comparable to optic neuritis is probably present either as an independent affection or in association with intracranial diseases like tumor and meningitis which exert pressure, cause irritation, or are accompanied by toxic agents which poison the blood and the lymph system. In chronic inflammation and other affections of the mucous membrane of the nose the olfactory nervules sometimes become involved in the inflammatory process. While olfactory neuritis is probably of more frequent occurrence than is usually supposed, it has been demonstrated in a very limited number of cases: the only case of which I know is the one reported by Althaus, to which reference is made in discussing primary degeneration of the olfactory nerve. Chronic olfactory neuritis is of comparatively frequent occurrence in syphilitic subjects, who often lose the sense of smell. Although toxic affections of the olfactory nerve, like similar disorders of the optic nerve, occur with considerable frequency, they are often overlooked because of the relatively small importance of the sense of smell as compared with that of sight. In olfactory neuritis the first stage is one of hyperesthesia, which, if the affection persists, in the course of days or weeks merges into anesthesia of the sense of smell.

Congenital Absence of the Olfactory Apparatus. The interior olfactory apparatus is sometimes congenitally absent or largely deficient. One of the most interesting cases in literature is reported by Claude Bernard, who found accidentally on a postmortem table that the intracranial olfactory apparatus was absent and that the cribriform plate of the ethmoid bone contained no foramina. Efforts were made to discover whether this woman had had the sense of smell, apparently with the result that she had. She was said to have had a great dislike to tobacco, was a good cook, was fond of flowers, disliked medicines with a nauseous flavor, and complained of some disagreeable odors. Still the testimony as to her preservation of smell was not of a convincing character. Such smell as she appeared to have had may have been simply the recognition of pungent and other substances which could be appreciated through the fifth nerve. Breschet has reported a case of congenital absence of the sense of smell which ran through several generations of the same family.

Primary Atrophy of the Olfactory Nerve and Tract. In tabetic cases gradual loss of smell is sometimes observed, and it progresses in almost the same manner as the loss of sight which is due to atrophy

of the optic nerve. A number of cases of this kind have been put on record, and it is what would be expected from the close analogies between the olfactory and the optic apparatus, and between both and the posterior columns of the cord. Althaus has recorded a case of tabes in which the patient had all the typical symptoms of this disease, and in which, very early in the development of the affection, the patient exhibited decided olfactory phenomena, followed eventually by complete loss of the sense of smell and of flavor. He suggests that the hyperosmia or hyperesthesia of the olfactory nerve present at first probably indicates a primary neuritis at this stage, this having been followed by secondary atrophy. Autopsy showed the naked eye appearances of a neuritis involving the olfactory bulb and olfactory nerve filaments at the base of the brain. Loss of smell in the aged or in those who are prematurely senile is sometimes due to atrophy of the olfactory nerve. The mucous membrane lining the olfactory channel differs from that of the respiratory passages. The latter is covered simply by the Schneiderian membrane, while the other has in it also pigmented cells which give to the membrane a brownish-yellow color. These represent a higher development of epithelial cells and of the olfactory end organs. A few curious cases are reported by Althaus which have an interesting bearing on the question of pigmentation and its relations to the functions of the olfactory nerve. In the case of a negro reported by Dr. Hutchinson, of Kentucky, the boy, affected with loss of pigment from the skin, gradually lost his sense of smell. The anosmia was probably due to the loss of pigment in the olfactory end apparatus. In an albino who had never had a keen sense of smell even this gradually failed, and disappeared entirely at about the age of sixty-three.

Secondary Olfactory Atrophy. Neuritis with subsequent atrophy of the olfactory nerve probably sometimes occurs in cases of intracranial tumors which are situated at points removed from the olfactory apparatus, the mechanism of the production of atrophy in these cases probably being similar to that which leads to both optic and auditory neuritis and atrophy in encephalic neoplasms which do not directly invade the optic or the auditory apparatus. Bull has recorded an interesting case which seems to bear out this explanation unless the olfactory atrophy was a mere coincidence. This patient, who had a syphilitic history, suffered from the general symptoms of brain tumor, and in addition had hemianopsia at first and later complete loss of sight, with atrophy of both disks. During the progress of the affection he lost the sense of smell and apparently with suddenness. Autopsy showed a small-celled sarcoma about the size of a large olive in the cuneus near the base of the occipital lobe on the left side. The olfactory lobes were atrophied, and the olfactory nerves reduced to mere threads.

Affections of Smell of Hysterical Origin. Unilateral loss of smell,

like other unilateral stigmata, is sometimes present in cases of grave hysteria, and, as a rule, on the same side as the hemianesthesia, hemiparesis, and other hysterical phenomena. Ferrier believed that a close examination of such cases would show that the effect is due to an anesthetic condition of the nostril and therefore is a phenomenon of general sensibility rather than of olfactory sensation. From an analogy with other hysterical phenomena, no good reason exists for supposing that the olfactory sense is not involved, although it is usually not entirely abolished, but simply in abeyance. In hysteria the sense of smell, like that of taste, may be completely obtunded; it may be hyperacute, or it may show peculiar perversions. To some individuals of the hysterical temperament certain smells are almost unendurable, and these may be odors which to others are particularly pleasant. In like manner certain articles of food or drink may be the source of great discomfort or absolute suffering.

Olfactory Nasal Neuroses. The so-called nasal neuroses of reflex origin have received much attention, and have perhaps been given a position of too much etiological importance by rhinologists and laryngologists in recent years. Their consideration properly belongs to affections of the trigeminal nerve and general and reflex nervous disorders, as they are usually attributed to a neurotic predisposition and a peripheral irritation of branches of the fifth nerve. They are especially observed in such affections as polypi, hypertrophic rhinitis, nasal stenosis, and ulcerations. While the nerve of common sensibility is by far the most frequently affected with such disorders, in comparatively rare instances irritation of the peripheral olfactory apparatus may be their source. It has been observed by some physiologists that various reflex phenomena, such as drawing up of the nares, arrest of respiration or of the heart's action, and slackening of the pulse, can be produced by acting upon the olfactory nerves, although they are undoubtedly, as a rule, dependent upon trigeminal irritation. Study and observation lead to the conclusion that in the presence of an unusual sensitiveness of the olfactory apparatus, and in neurotic or neurasthenic patients, olfactory irritation may occasionally give rise to reflex nervous disturbances. Clinical observations in support of this view are, however, almost entirely wanting. Joal has called attention to the many cases recorded in medical literature of severe headaches and of nervous disturbances, and even occasional cases of death, due to the inhalation of odors of flowers. A number of actors and singers suffer in this way, and generally it is a certain odor which cannot be tolerated. The symptoms set up are usually coryza, hoarseness even to aphonia, and headache.

Etiology.—The majority of cases of loss of smell are due to disease of the nasal mucous membrane, which may be associated with or lead to disease of the peripheral sense organ. Falls, blows, or other injuries may cause peripheral olfactory disease. Affections of

smell of cerebral origin may also originate from injuries. Cases have been reported in which after injury to the head all substances acquired some peculiar smell or taste, and in other instances the sense has been gradually impaired or lost. A form of intermittent anosmia has been described as occurring in the course of malaria. Traumatic anosmia is not infrequently caused by blows on the occiput by a form of *contrecoup.* Disease of the fifth nerve may be indirectly the cause of anosmia, lesion of this nerve leading to trophic changes in the mucous membrane and other tissues of the nose, and in this way to destruction of the olfactory cells and filaments. Excessive stimulation of the sense of smell, as by the persistent use of strong odors, may cause anosmia, which has been known to result from frequent and prolonged inhalation of ether or chlorine and from undue stimulation of the olfactory nerve by the electric current, even when no burning or destruction of tissues has been produced. In the treatment of nasal affections by douches or atomizers the too frequent use of strong solutions may cause anosmia. In cases of paralysis of the seventh nerve the sense of smell may be impaired because the patient is not able to sniff at the substances; but this is not likely to be noted when only one side of the face is paralyzed. Influenza, rheumatism, or any other infectious disease may lead to anosmia, through either its direct or its indirect effects upon the olfactory nerve. The most important intracranial causes of anosmia and other etiological factors in its production have been considered when discussing the varieties of olfactory disease.

Pathological Anatomy.—Evidences of inflammation, of atrophy, primary and secondary, of hypertrophy, of new growths, or of other focal lesions, are usually present somewhere in the olfactory path. In a few cases, when the disease is apparently of functional or hysterical origin, it passes away, leaving no trace behind. From reported cases it would seem that most commonly the loss of smell when of cerebral origin is on the side of the lesion.

Diagnosis.—The existence of anosmia is to be determined by a careful investigation of the patient's sense of smell, a test which is often overlooked by the clinician. Each nostril should be tested separately. We may eventually learn to distinguish anosmia due to disease of the anterior and of the posterior nares, or even when the lesion is in different areas within the nasal cavities. Separate examinations should be made of the senses of smell, of taste, and of general sensibility. In every case of disorder of smell a thorough local inspection of the throat and nose should be made. Occasionally intracranial disorders are suspected when the local causes are quite sufficient to explain the affection. Galvanic stimulation of the olfactory nerve causes peculiar olfactory reactions, as a phosphorus-like odor. This fact may be made use of to determine the presence or absence of true olfactory nerve disease. If no response can be elicited, it may be

regarded as certain that the nerve is seriously involved. The method of making this examination is to place one electrode in a weak saline solution with which the nose is filled, and carefully increase the strength of the current. If great care is not taken, vertigo and injury to the retina may be caused. The symptoms which accompany the anosmia or hyperosmia will sometimes indicate the position and ramifications of the lesion. Optic neuritis is usually present, but, as this may accompany unilateral growths even when they are not situated in the prefrontal region, the symptom is not helpful in the accurate local diagnosis of olfactory affections. Some form of temporal or nasal hemianopsia may be determinable in cases in which the olfactory bulb and roots are directly involved; at least this may be done before optic neuritis and atrophy have destroyed the patient's sight. The special physical symptoms of tumors of the prefrontal lobe are found present in cases with anosmia, and in some instances motor aphasia and motor agraphia may indicate direct and indirect involvement of the centres for motor speech and writing. Symptoms showing inflammation of the hypophysis and of the nerves of the ocular muscles may be present.

Prognosis.—Speaking generally, the prognosis of disease of the olfactory apparatus is bad. It is that of the various lesions which give rise to such disease,—of intracranial tumor, meningitis, encephalitis, abscess, hemorrhage, softening or aneurism; of olfactory neuritis; of primary degeneration of the nerve, and of hysteria. Diseases of the nasal mucous membrane, if long continued, usually cause permanent impairment of the olfactory nerve. A few cases of traumatic or syphilitic origin recover under proper treatment, and disorders of smell due to hysteria may disappear like other hysterical stigmata.

Treatment.—When syphilis is known to be present, or even when it is suspected, the patient should be placed upon a proper course of mercurials or iodides or on the mixed treatment. Snuffs to stimulate the nerve locally may be employed, and among these one which has frequently been suggested is composed of strychnine sulphate in olive oil (a one per cent. solution) or mixed with sugar or powdered gum arabic (one thirtieth of a grain to two drachms). When galvanism is employed, a weak current should be used, putting one electrode, preferably the cathode, to the nasal bone, and the other to the back of the neck or to some other indifferent spot. Hirt noticed that during the passage of a galvanic current from one side of the head to the other a patient experienced a constant odor of the oil of lavender, showing that it is not impossible to affect the cerebral centres for smell by external electrical treatment. When an affection of smell is due to a tumor, abscess, hemorrhage, or other focal lesion invading the olfactory apparatus, the treatment should be based on the principles that apply to such lesions wherever situated.

AFFECTIONS OF TASTE DUE TO DISEASE OF THE NERVES OF TASTE AND OF THEIR RELATED ENCEPHALIC STRUCTURES.

Essential and Accessory Nerves of Taste.—The essential nerves of taste are those which are indispensable to the gustatory sense; the accessory nerves of taste are those concerned with the regulation of the movements necessary to the correct appreciation of taste. The integrity of the chorda tympani and of the glossopharyngeal nerve is essential for the complete performance of the gustatory function. The hypoglossal is the chief motor nerve to the tongue, but the facial probably gives some motor branches to this organ. The pneumogastric supply is by way of filaments which pass through the superior laryngeal nerve to the posterior part of the tongue, and it is by means of these branches that a reflex connection is established between the tongue and the stomach, this playing a most important rôle in the act of swallowing. As the soft palate, palatal arches, hard palate, cheeks, epiglottis, larynx, and possibly the lips, have taste buds distributed in them, the nerves which supply the muscles contained in these parts—motor branches of the glossopharyngeal, the inferior or recurrent laryngeal, and the facial—may also be regarded as accessory nerves of taste, as they assist in the full performance of the complicated processes connected with the exercise of this sense. Our concern in this section will be almost entirely with the essential nerves of taste.

Peripheral Gustatory End Organs.—The peripheral end organs of the nerve of taste are known as taste buds or taste bulbs. These are oval or flask-shaped bodies found in the epithelial lining of the tongue, being present in comparative abundance in the epithelium of the circumvallate papillæ. They are also found on some of the fungiform papillæ and in the folds in the neighborhood of both the circumvallate and the fungiform papillæ. Special groups of parallel folds known as the *papillæ foliatæ*, which are situated in the sides of the tongue near the anterior pillars of the fauces, and which are much more highly developed in some of the lower animals and especially in the rabbit, contain a considerable number of taste buds. Taste buds are also distributed, as stated in the last paragraph, in the mucous membrane of the soft palate, the palatal arches, the hard palate, the cheeks in rare instances, the epiglottis, the larynx, and, according to some authorities, in the lips. They are enclosed in a superficial tegmental or cortical layer of elongated epithelial cells. At the superficial pole of the taste bud is a minute canal, the taste pore, which connects the interior of the bud with the mucous membrane. The so-called gustatory cells have a nucleated, somewhat bulging body, with a distal or peripheral and a proximal or central process. The peripheral process ends in a taste hair, which passes into the gustatory pore. In Fig. 26, page 21, is shown a diagram represent-

ing a unipolar nerve cell of one of the ganglia of the nerves of taste (geniculate, petrous, or jugular ganglion), one of whose bifurcations surrounds and comes in contact by its terminals with the gustatory end organ. These so-called gustatory cells are not true "nerve cells," but are modified epithelial cells which disappear or undergo great change after section of the nerves of taste, but do not undergo true degeneration. They are transformed into ordinary epithelial cells. The influence determining the function and the form of the cell being withdrawn, the cell reverts to its ordinary type. The union of the nerve with its sensory epithelium has been determined to be indirect, that is, by contact, trophic cells being more centrally located. This is true at least for the tactile, gustatory, and acoustic apparatuses.

Areas in which Taste is normally appreciated.—My own investigations, with the experiments of Frankl-Hochwart and the observations collected by this writer especially from German sources, lead to the following conclusions regarding the normal distribution of the taste sense. The appreciation of the taste of different substances varies somewhat in different portions of the tongue, even in the same individual, and the variations among different persons are considerable. Taste is acute at the tip of the tongue, both on its upper and its lower surface; passing backward, in the anterior two-thirds of the dorsum of the tongue it is more acute towards its lateral borders. Near the raphe in the middle portion of the anterior two-thirds of the tongue, taste is sometimes not present or is poorly developed. Taste for certain substances may be present, or even acute, in this locality, while it will be absent for other substances; thus, in one of my own cases it was present for acids like citric acid, but not for saline substances, while in another it was present for salt alone. The sense of taste is very acute in and around the circumvallate papillæ. Bitter substances seem to be best appreciated by the posterior portion of the tongue. The greatest uniformity as to the acuity of taste for all substances is shown by that portion of the tongue at and behind the circumvallate papillæ, and by the sides and the tip of the tongue both on its upper and its under surface. The variations in the acuity of taste appear to be greater in the distribution of the chorda tympani than in that of the glossopharyngeal nerve. On the dorsum of the tongue, in front of the region of the circumvallate papillæ, except at the extreme tip, and also on the under surface of the tongue in a strip along the raphe, salt may be better appreciated by one, and sweet or acid substances by others. The degree of the appreciation of each or of all may vary. Taste is sometimes acutely present in the region of the hard palate, an observation denied by some. In nearly all the cases examined by me salt was appreciated on the hard palate. Frankl-Hochwart found taste present on the pillars of the fauces in five out of eight cases tested

by him. The mucous membrane of the cheeks in most adults is not a taste area, although some children have taste in this region. One of my subjects could taste molasses applied to the mucous membrane of the cheeks, but could not here appreciate other substances, even sugar. The areas for taste, in the order of their importance, are, according to Frankl-Hochwart, the posterior third of the tongue, the borders and tip of the tongue, the soft palate, the uvula, the anterior palatal arches, the posterior pillars of the fauces, the tonsils, the posterior wall of the pharynx, the middle of the tongue, the lower portion of the tongue, the hard palate, and the interior of the larynx. While accepting this statement as giving nearly the correct order for most persons, I should be inclined to doubt the position assigned to two or three of these areas. In the hard palate and a portion of the under surface of the tongue, taste is certainly acute in some individuals, and the mucous membrane of the cheeks should be added for some children and a very few adults.

Delicacy of Taste.—Bailey and Nichols, as the result of their examination of eighty-two males and forty-six females, reached several interesting conclusions as to the delicacy of taste. These were that the sense of taste is far more delicate for bitter than for other sapid substances; that the order of delicacy is, bitter, acid, salt, sweet, and alkaline; and that the sense is more delicate in women than in men, an apparent exception to this being shown in the case of salt. Solutions of quinine, sulphuric acid, common salt, sugar, and sodium bicarbonate were used in testing.

Electrical Taste.—What is sometimes called electrical taste—the taste produced by the application of a weak galvanic current to the tongue—is usually designated as acid when the anode is used for testing, and as alkaline when the cathode is used. Frankl-Hochwart experienced a burning or sticking sensation at the cathode, and Volta obtained an alkaline taste, while others have described it as bitter or acid. This electrical response is not a pure taste phenomenon, but a combination of sensations. It is an old and not yet finally decided question whether the gustatory reaction is due to a direct irritation of the taste nerves by means of the electric current or is dependent upon electrolysis of the fluids of the mouth.

The Course and Origin of the Nerves of Taste.—Both the peripheral and the central course of the nerve or nerves of taste, and the position of their nuclei of origin, have been subjects of much dispute. On general principles it is most probable that taste, like the other special senses, is subserved by only one nerve, although this may be subdivided in its course from periphery to centre. It is also most probable that this nerve is the glossopharyngeal. The chorda tympani nerve was once regarded as a true branch of the facial, but it is simply in contact with the facial in a part of its course. A second view, and one which is upheld by many authori-

ties, is that it is a branch of the fifth nerve. The most probable view is that it is a peripheral continuation of the intermediary nerve of Wrisberg, and, as this terminates very close to the sensory nucleus of the glossopharyngeal, it may be regarded as an aberrant fasciculus of this nerve. Many facts of physiology and clinical medicine show that the chorda tympani plays an indisputable rôle in the function of taste; and the chief points in dispute are as to its course and mode of origin.

Physiological and Clinical Evidence that the Chorda Tympani is one of the Nerves of Taste.—Division, either experimentally or by disease, of the chorda tympani in its course with the facial invariably causes loss of taste on the anterior portion of the tongue on one side. Perforations of the membrana tympani and disease and operations on the ear have led to the same result. Cases have been recorded in which, after such lesion, the application of stimuli to the distal portion of the chorda tympani has caused sensations of taste in the region in which it was lost or impaired. In a case recorded by Shulte of deafness with otorrhea and polypus, in removing the latter the chorda tympani was severed. The patient immediately experienced a sensation as if a membrane was stretched from the left side of her tongue, and at the same time taste was completely lost on this side from the tip back to a point where the circumvallate papillæ commenced; but on the posterior third of the tongue taste was not impaired. Lesions of the facial nerve outside of the stylomastoid foramen, and lesions within the cranium, if strictly confined to this nerve, do not cause impairment or destruction of taste on the tip of the tongue. These cases clearly prove that lesion of the chorda tympani in its course with the facial causes interference with taste. The points in dispute relate rather to the central connections of the chorda tympani and its course before joining the facial. Vulpian and other physiologists have found degenerated fibres in the tongue after section of the chorda tympani. While cases have been recorded in which, after excision of the lingual branch of the fifth, complete loss of both sensibility and taste on the corresponding side and tip of the tongue has occurred, Schiff has shown that if this nerve is divided before it receives the chorda tympani from the facial, loss of taste does not result.

The Gustatory Division of the Glossopharyngeal Nerve.—*Origin, Root Fibres, and Oblongatal Nuclei.* The glossopharyngeal nerve is usually regarded as springing from three regions, two of which at least are distinct cell nests. One of these two gives origin to the motor portion of the nerve, with which we are not at this time specially concerned; the other is the terminus of the gustatory fibres. The sensory (gustatory) nucleus, sometimes spoken of as the sensory glossopharyngeal nucleus, contains small cells, the motor (nucleus ambiguus) large cells. As of course the gustatory subdivision is an

afferent nerve, its true origin is peripheral. In the jugular foramen, as has already been stated, two ganglia, the petrous and the jugular, the real origins of the nerve, are found upon the trunk of the nerve, these corresponding to the dorsal spinal ganglia, and from them the processes which together constitute the nerve pass both peripherally and centrally. From the so-called superficial origin of the nerve its rootlets pass mesad and dorsad through the bulb to the special nucleus beneath the inferior fovea in the floor of the fourth ventricle; some of the fibres turn downward into the fasciculus solitarius, and others, probably efferent, bend forward to the upper prolongation of the accessory vagal nucleus (nucleus ambiguus). Ramón y Cajal in his recent work on the oblongata, after corroborating the most important results of Retzius, His, Martin, Edinger, Held, and Koelliker, has added some important facts with regard to the course, methods of contact, and termination of the root fibres of the glossopharyngeal nerve. He concludes that the common sensory vago-glossopharyngeal roots end in two distinct gray masses by means of collaterals in the superior and in the descending nucleus, which in reality form a single nucleus, by means of end ramifications in the *commissural nucleus*. These end ramifications cross in great part the middle line, and form a true decussation of the sensory root. The location of this commissural nucleus is a new observation by this investigator. It consists of fasciculi which, close under the ependyma of the ventricular floor, approach the raphe, their gray terminal masses there uniting. Three-fourths of the fibres of the fasciculus solitarius terminate decussating in this nucleus or ganglion. It is an oval, somewhat arched nucleus between the ependyma and the most external fibres of the gray commissure of the cervical cord. According to Obersteiner, the gray matter of the fasciculus solitarius (that is, of the vertical nucleus) is very similar to the gelatinous substance of the spinal root of the fifth, and he holds that it must be considered very doubtful whether vagus fibres unite with the fasciculus solitarius. At all events, these fibres cannot be numerous, and the chief mass of them comes from the nervus glossopharyngeus.

Peripheral Course. The nerve has its so-called "superficial origin" in the groove between the olivary and the restiform body by five or six filaments arranged in a vertical line commencing immediately below the facial nerve. Traced backward from this point, it proceeds outward, somewhat forward, close to the flocculus, to the jugular foramen, through which it passes with the pneumogastric and spinal accessory nerves. After emerging from the jugular foramen it bends forward and inward between the internal jugular vein and the internal carotid artery, under the stylopharyngeus muscle. Winding around this muscle, it passes upward and forward to reach the hyoglossus, where it spreads into its lingual branches. The gustatory fibres begin in the taste buds, which are distributed to the

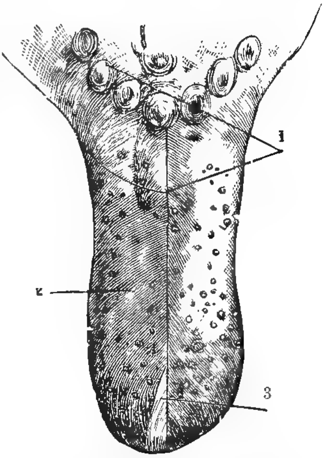
posterior part of the tongue, to the soft palate, to the palatal arches, to the epiglottis, and possibly to other structures.

The Nerve of Jacobson. The nerve of Jacobson, or tympanic branch of the glossopharyngeus nerve, passes from the petrous ganglion through the tympanic canaliculus to the tympanic plexus. From the tympanic plexus are given off numerous fine branches. Among the most important of these are communicating branches to the greater and to the lesser superficial petrosal nerve. Others pass to the Eustachian mucous membrane, to the fenestra ovalis, to the fenestra rotunda, and to the gangliated (sympathetic) system. Some branches of communication between the glossopharyngeal and the vagus, sympathetic, and facial also spring from the petrous ganglion.

Evidence that the Glossopharyngeal is the Main Nerve of Taste.—Pope has recorded a case which proves that the glossopharyngeal is a true nerve of taste. A thrombus of the left vertebral artery constituted a tumor which made pressure on the roots of the glossopharyngeal nerve. The only other nerve involved was the pneumogastric,—the facial, the fifth, and the hypoglossal being out of the reach of pressure. Acid and sweet solutions were used in testing, and the tests were made with great care. On the posterior left side of the tongue neither solution could be tasted. On the tip of the tongue, on the same side, the acid was tasted but the sweet solution was not perceived. Other symptoms present were diminution of power in the left arm, hand, and leg; inability to swallow either liquids or solids; slight want of expression on the left side of the face; contraction and insensibility to light of the left pupil; and some impairment of tactile sensation. This case seems to prove conclusively the part played by the glossopharyngeal in the function of taste. In a case reported by Lehmann, in which, however, no post-mortem examination was made, basal fracture caused paralysis of the facial, auditory, hypoglossal, and vagus nerves on the right side; on which side, throughout the length of the tongue, taste was abolished, except at the point, where it remained in very slight degree. This case also bears out the views here advanced, as the pars intermedia of Wrisberg and the main glossopharyngeal would in all probability be also involved in a lesion implicating the other nerves named. Rosenberg reports that after section of the glossopharyngeal nerve in the neck of a rabbit the taste buds disappear in the circumvallate papillæ and the papillæ foliatæ of the same side, as has been described by Vintschgau and Hönigschmied. Baginsky had stated as the result of prior experiments that the taste buds persisted after section of the glossopharyngeus, but Rosenberg flatly contradicts his statements. Numerous taste buds are found in the papillæ of the anterior third of the tongue after section of the glossopharyngeus. After cutting the lingual nerve in the neck (the section probably including the chorda tympani), the taste buds in the anterior third of

the tongue degenerate (or involute, probably reverting to ordinary epithelial cells) in great numbers, while those in the circumvallate papillæ and the papillæ foliatæ remain unchanged. Even after section of the lingualis scattered taste buds remain at the point of the tongue, which seems to signify that the taste fibres for the anterior portion of the tongue are not all contained in the chorda tympani nerve. It may be that some of them are connected with the glossopharyngeal nerve, as Hirschfeld's branch of the glossopharyngeus may be traced in the tongue almost to the point. In a case in which Walsh stretched the lingual nerve for a severe neuralgia of the tongue of long standing, taste was lost over fully three fourths of the dorsum and the side of the right half of the tongue, with the exception of a small area near the tip of the tongue, in which only slight loss of taste was present; over the posterior one-fourth of the tongue taste was present, but was scarcely more than perceptible except when dilute acetic acid was placed directly upon the circumvallate papillæ, when taste seemed quite active. The under surface of the tongue was devoid of taste. In this case the preservation of taste in the small area near the tip of the tongue (Fig. 345) might be accounted for by the fact that this area is probably supplied by Hirschfeld's branch of the glossopharyngeus. The parts in which taste was affected were those which are supplied by the chorda tympani, which of course runs with the lingual in the tongue. As the result of numerous examinations of individuals of different ages with presumably normal taste, and also of the study of several old and recent cases of facial paralysis with loss of taste in the distribution of the chorda tympani nerve, the diagrams of the normal distribution of the gustatory fibres of the glossopharyngeal and chorda tympani nerves to the tongue have been made. (Fig. 346.) These show that the glossopharyngeal nerve is distributed to the posterior portion of the dorsum of the tongue from the circumvallate papillæ backward, and also to a strip of the lateral aspect of the tongue and to a small area at its tip. The probable distribution of the chorda tympani nerve in man is, as shown, to the dorsum of the anterior two thirds of the tongue, to its lateral edge, and, on the under surface of the tongue, to a strip bordering the raphe on each side from the frenum to the tip. In the cases examined, taste seemed to be normally absent in the central portions of the under

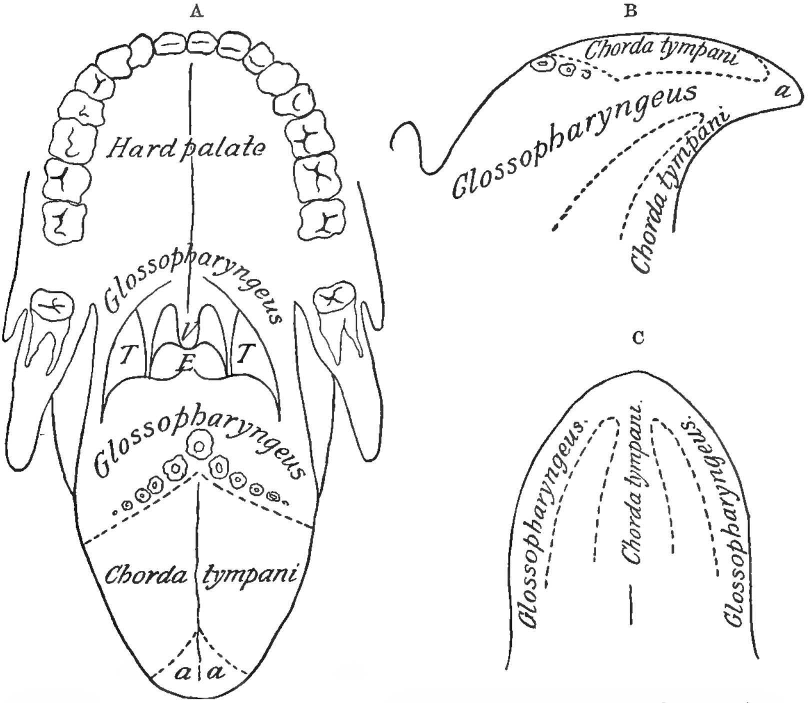
FIG. 345.



Effect on taste and common sensibility of stretching of the lingual branch of the fifth nerve: 1, area of diminished sensibility; 2, area of absolute loss of sensibility and sense of taste; 3, area of diminished sensibility and sense of taste. (Walsh.)

surface of the tongue, with the exception of the narrow strip just described, along the raphe. (See diagram, Fig. 346, C.) It is not improbable that both glossopharyngeal and chorda tympani fibres are commingled in the small area *a*, Fig. 346, B. Rosenberg states that he has had the opportunity to examine the circumvallate papillæ of a man in whom the glossopharyngeus of one side was completely degenerated on account of a neoplasm at the base of the skull. On

FIG. 346.



Diagrams showing the usual distribution of the glossopharyngeal and chorda tympani nerves on the dorsum, sides, and under surface of the tongue and the hard palate, based upon a study of individuals in whom taste was normal, and of cases in which the chorda tympani was the seat of lesion. A, dorsum of the tongue and the hard palate; *E*, epiglottis; *T*, *T*, tonsils; *V*, uvula; *aa*, area supplied by Hirschfeld's branch of the glossopharyngeus (and possibly also by some fibres from the chorda tympani). B, side view of the tongue; *a*, area supplied by Hirschfeld's branch of the glossopharyngeus (and possibly also by some fibres from the chorda tympani). C, under surface of the tongue.

the side of the degeneration he found only two taste buds in a number of sections; the others had disappeared, and their places were occupied by epithelial cells. Some evidence indicates that implication of the tympanic plexus and the nerve of Jacobson (tympanic branch of the glossopharyngeal) causes loss of taste on the posterior part of the tongue. If this be the case, the taste fibres may escape from the nerve of Jacobson and pass through the small superficial petrosal nerve and thence to the third branch of the fifth.

The Intermediary Nerve of Wrisberg.—*Origin.* Huguenin and Krause supposed that this nerve arose in the ventral auditory nucleus, but the observations of Martin and His showed that the fibres arise in the geniculate ganglion. They were traced by His into the "solitary bundle." Duval considered the pars intermedia in a certain measure merely the superior part of the sensory glossopharyngeus roots, but, according to Koelliker, Duval does not mean by the sensory glossopharyngeal roots the fibres of the ninth nerve which enter into the fasciculus solitarius, but those which unite with the vagoglossopharyngeal end nucleus on the floor of the fourth ventricle laterad from the hypoglossal nucleus. Koelliker, with Martin and His, believes that this glossopharyngeal sensory nucleus is the nucleus of the fasciculus solitarius, the terminal nucleus of the descending root of the vagoglossopharyngeal. The bundles of the intermediary nerve of Wrisberg either pass through the spinal root of the fifth or lie between it and the vestibular nerve. With regard to the nuclear relations of the pars intermedia, Obersteiner states that in the portion of the vertical nucleus of the glossopharyngeus which extends cephalad of the point of bending of the solitary bundle (descending root of the glossopharyngeus) root fibres enter which no longer belong to the glossopharyngeus, but to the intermediary nerve of Wrisberg. According to Obersteiner, this nerve arises in the geniculate ganglion, which has the same construction as the spinal ganglia, and into which the chorda tympani as a peripheral nerve enters. With regard to the homology between the geniculate ganglion and the spinal ganglia, it may be remarked that Retzius, in 1880, found, in the cat, dog, and man, just such multipolar cells in this ganglion as are present in the spinal ganglia. This is in harmony with the results of Martin and His, who in the human and the cat embryo have found bipolar cells in the geniculate ganglion the same as in the spinal ganglia. The central branches of the axis cylinders of the cells of the geniculate ganglion were found by Lenhossék to be almost always smaller than the peripheral, corresponding in this to the spinal ganglia. The peripheral processes appeared to join the facialis. Lenhossék could not determine the course of all the axis cylinders of these cells. The central fibres unite in a distinct bundle and form the intermediary nerve. Lenhossék emphasizes the great similarity between the formation of the facialis and intermediary nerve and that of a pair of spinal roots, referring to the junction (or juxtaposition) of a motor nerve with the ganglion and the mingling of both motor and sensory fibres on the far side of the ganglion. The facial and chorda tympani have a segmental companionship similar to that which exists between the motor and the sensory spinal nerves. It is regarded by Lenhossék as most probable that the fibres of the intermediary nerve which pass from the ganglion into the facialis form the chorda tympani, although he admits

two other possibilities: a portion of these sensory fibres may remain within the facialis as far as its ramifications in the face, it being well known that the facial nerve is sensitive immediately on leaving the stylomastoid foramen, or some of the fibres at the crossing of the facialis with the auricularis vagi may pass into the latter.

FIG. 347.

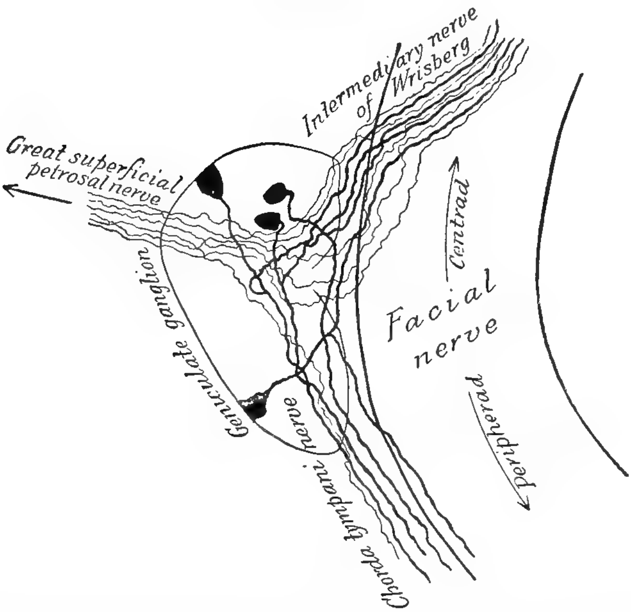


Diagram of the geniculate ganglion, showing the central and peripheral processes of its ganglion cells, and other nerve processes which pass through it. The intermediary nerve of Wrisberg is seen to be constituted by some of the central processes of this ganglion, and the chorda tympani nerve by some of its peripheral processes; fibres of the great superficial petrosal nerve pass through the ganglion, while the facial nerve passes by the ganglion. (Modified from Lenhossék.)

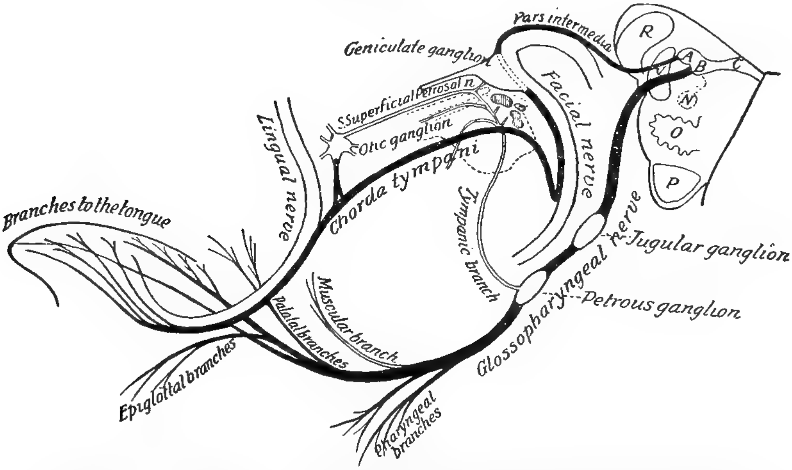
Peripheral Course. The intermediary nerve of Wrisberg, according to Duval, arises* at the upper or cephalic extremity of the sensory nucleus of the glossopharyngeal nerve, and, passing obliquely through the pons, its fibres emerge between the facial and auditory nerves at their superficial origins. It then takes its course as a small, compact, independent bundle to the internal auditory meatus, which it enters, running dorsally to the auditory nerve and still between it and the facial. It joins the facial bundle at the geniculate ganglion.

Physiological Views. Some writers have held that the chorda tym-

* In discussing and describing the cranial nerves it must be remembered that the expressions used by the older anatomists and especially by the authorities on gross anatomy usually have reference simply to the position and relations of these structures in dissections. From the embryological and physiological points of view the afferent or sensory nerves terminate rather than rise in the bulbar and spinal nuclei. The nuclei of these nerves so often spoken of as nuclei of origin are in reality terminal or end nuclei.

pani nerve was a distinct cranial nerve, believing it was a branch or continuation of the intermediary nerve of Wrisberg. Sapolini believed in the existence of a separate or thirteenth cranial nerve, of which the chorda tympani is the peripheral ending, and the pars intermedia of Wrisberg a central portion. He traced it from the fourth ventricle to the geniculate ganglion, and from the chorda tympani backward to the same ganglion, and believed these two portions to be one and the same nerve, running with the seventh, but anatomically distinct from it. Sapolini regarded it, however, not as the nerve of taste, but as the special nerve of speech. Penzo also held that the chorda tympani was a continuation of the pars intermedia. Bigelow arrives at the same conclusion, believing that the lingual branch of the fifth presides over common sensibility alone.

FIG. 348.



Peripheral gustatory apparatus: *A*, portion of the sensory glossopharyngeal nucleus in which the pars intermedia terminates; *B*, main portion of the sensory glossopharyngeal nucleus; *C*, commissural nucleus of Ramón y Cajal; *N*, nucleus ambiguus; *O*, olive; *P*, pyramid; *R*, restis; *V*, spinal root of the fifth nerve.

According to him, if the intermediary nerve of Wrisberg be cut in the aqueduct behind the ganglion taste will be destroyed. The pars intermedia is a smaller nerve than the chorda tympani as ordinarily understood; but it is known that both vasomotor and sensory fibres pass into the chorda tympani, and it is not impossible that it may contain some fibres of common sensibility derived from the fifth through the Vidian. In Fig. 348 is shown diagrammatically the entire peripheral apparatus of taste according to the views of the author (as presented in the preceding pages).

Supposed Relations of the Fifth Nerve to the Sense of Taste.—*Impairment of Taste coincident with Lesions of the Fifth Nerve.* The facts in favor of the fifth nerve being concerned with the function of taste are of a somewhat diverse character, and some of them

seem at first sight to be almost convincing, although the author's view is that the glossopharyngeal (including in this the intermediary nerve of Wrisberg) is the only essential nerve of taste. The facts and views of those advocating this fifth nerve hypothesis should, however, be presented, as the question is still regarded by many as unsettled. Cases, without autopsy, have been recorded in which disease of both motor and sensory subdivisions of the fifth between its superficial origin and the Gasserian ganglion have been associated with loss of taste. Several have fallen under the notice of the author. In some of these cases the loss of taste has been confined to the tip of the tongue; in most of them both the anterior and posterior portions of the tongue have been implicated. The clinical and pathological evidence shows clearly that the first or ophthalmic subdivision of the fifth nerve does not contain gustatory fibres. As to whether any run in the second or third branch the apparent evidence presented by those who believe that the gustatory fibres are contained in the fifth nerve is conflicting.* Schiff and Vulpian, in their experiments on animals, thought that section of the fifth caused loss of taste. Lussana also found that section of the lingual was followed by loss of taste on the anterior two-thirds of the same side. Frankl-Hochwart, who has contributed the most valuable of recent articles on taste to Nothnagel's "Specielle Pathologie und Therapie," believes that the fibres of taste are from the fifth nerve, and that it is not known whether the fibres are in the second or the third branch. A few of the operations for the removal of the Gasserian ganglion and section of the nerves cephalad of this ganglion seem to be in favor of the view that either the second or the third branch of the fifth nerve transmits gustatory fibres. In a case reported by Winslow taste was lost on the side of the operation. In one by Rose, in which Ferrier tested for taste, it was found to be completely lost on the affected side of the tongue. In a case of Stewart's, in which two operations were performed, one for the removal of Meckel's ganglion and the other for the removal of the Gasserian ganglion, taste was lost on the same

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* On this point William Aldren Turner, from whose paper on Facial Paralysis and the Sense of Taste (Edinburgh Hospital Reports, vol. iv., 1896) many interesting data have been obtained, speaks as follows: "There is much contradictory evidence as to which branch or branches of the fifth nerve transmit fibres of taste. The first or ophthalmic branch may with certainty be excluded. There are, on the other hand, numerous cases in which the distribution of anæsthesia indicated a lesion of the first and second divisions and an intact state of the third, where taste was abolished. On the other hand, there exist cases in which the anæsthetic area pointed to a lesion of the third division only, and in which taste also was lost (Romberg, Ziehl). Or, again, a case of Erb's showed paralysis of the third trigeminal branch, with an intact state of the first and second, and the retention of taste; while cases by Jaffe, Stamm, and Schmidt show retention of taste where the third branch was free, and the first and second nerve, either in whole or in part, were paralyzed."

side of the tongue; and Krause reports a similar case in which after entire removal of the Gasserian ganglion taste was dulled but not lost. In six out of seven cases reported by Tiffany, taste was carefully studied; in two taste was lost, as it was also in one of two cases recorded by Finney. A case reported by Ferguson seems to prove the passage of the gustatory fibres through the fifth nerve. This case is certainly most difficult to explain except on the theory that the fifth nerve is concerned with taste. The only explanation that can be offered in opposition is the possible existence of a second unnoted lesion of the pars intermedia of Wrisberg. The patient had complete loss of taste on the tip and left side of the tongue, the posterior part of the tongue, the fauces, and the palate retaining the sense of taste. Autopsy showed a small exostosis in the scaphoid fossa pressing on the posterior opening of the Vidian canal and by pressure destroying the Vidian nerve. The nerve degeneration was traced by Ferguson along the main course of the Vidian, and thence along its two branches, the carotid and the petrosus major, which enters the geniculate ganglion of the facial. The degeneration could be readily followed until the point was reached at which the chorda tympani was given off, and from this point it followed the chorda tympani to the lingual branch of the third division of the fifth nerve, and thence along the lingual. Ferguson argued from this case that the course of the nerves concerned with taste must be from the superior maxillary nerve into the sphenopalatine ganglion, thence through the Vidian canal to the gangliform enlargement of the facial, along this to the chorda tympani, and through the chorda tympani into the lingual.

Manner in which the Chorda Tympani is supposed to pass in the Trigeminus. Schiff and Erb believe that the chorda tympani joins the fifth nerve by way of the greater superficial petrosal nerve, the Vidian nerve, and the sphenopalatine ganglion. Frankl-Hochwart suggests that possibly a few fibres which do not mingle with the chorda tympani leave the lingual nerve more centrally, and pass by way of the otic ganglion, and by other tracts, as yet unknown, to the branch of the fifth. Ziehl regards the lesser superficial petrosal nerve as the one which connects the chorda tympani with the fifth nerve. Other hypotheses, more or less strained, have been suggested to explain the manner in which the chorda tympani is distributed centrally with the fifth nerve; but I believe that the facts and views here advanced are convincing against this widely accepted theory. It is not improbable that some fibres pass from the pars intermedia of Wrisberg by way of the geniculate ganglion and greater superficial petrosal nerve to the sphenopalatine ganglion, and thence with the palatine nerves to the hard palate. The hard palate is supplied with gustatory fibres either in this way or by a greater extension forward of the palatal branches of the glossopharyngeus than is usually described.

Facts opposed to the View that Gustatory Fibres are contained in the Fifth Nerve. Satisfactory explanations can be offered for most, if not for all, of the facts just cited, without accepting the view that the fifth nerve contains taste fibres. In regard to the experiments of Schiff and Vulpian on the lingual nerve, it is to be noted that the section may involve the chorda tympani after its union with the lingual,* although Schiff and Ferrier have reported instances in which partial loss of taste seemed to have resulted from severance of the lingual before its junction with the chorda tympani. With regard to the cases of lesion at the base of the brain involving the fifth nerve, as clearly shown by the clinical phenomena and postmortem evidence, it must be remembered that the lesions may also have involved the gustatory tracts, passing in the pons from the oblongata to the cortex, or in some cases even the glossopharyngeal or pars intermedia of Wrisberg at or near the brain stem. It needs also to be recalled in this connection that the root bundles of the nerve of Wrisberg pass, as indicated by Koelliker, either through the spinal root of the fifth or between it and the vestibular nerve. In this way symptoms chiefly referable to the fifth and the pars intermedia may sometimes originate and be supposed to belong only to the fifth. Bruns has reported a case in which a lesion of the root of the fifth nerve occurred without loss of taste. Owing to the connection which exists between the Vidian nerve and the sphenopalatine or Meckel's ganglion, it would be expected that excision of this structure would cause loss of taste on the anterior two thirds of the tongue; but such was not the case, according to the observations of Bastian and Ferrier. While, as stated, some of the Gasserian ganglion operations would seem to be in favor of the gustatory functions of a portion of the fifth nerve, the results of removal of this ganglion and of cutting the nerves cephalad of it are on the whole opposed to the view that the proximal continuation of the chorda tympani is in the fifth nerve. It is true that in some of the cases the records report blunting or loss of taste in the tongue, usually in its anterior portion, on the side on which the operation has been performed; but this impairment can be accounted for in several ways. In Horsley's operation by raising the temporal lobe it is possible to injure the cortical tracts and centres for taste. In an operation so serious and extensive as that for the removal of the Gasserian ganglion other parts may be injured. The perversion of common sensibility brought about by the fifth nerve operation might in some instances be confounded with blunting of taste, although this is not probable with careful observers. According to Bechterew, general facial anesthesia may be accompanied

* The term *gustatory* as applied to the lingual branch of the fifth nerve should be dropped, as, to say the least, the gustatory functions of this nerve are doubtful.

by impairment of the special senses without involvement of their nerves. He has recorded a case of gunshot wound of the cervical region and the lower part of the oblongata, which caused on the entire left side of the body, with the exception of the face, temporary paresis with anesthesia for pain and temperature and considerable impairment of touch and pressure with preserved muscular sense; on the entire right side of the body, with the exception of the face, at first complete paralysis, and later paresis, great impairment of sensibility to touch and pressure; and loss of muscular sense and anesthesia for all forms of sensation on the right side of the face. In this case notable impairment of sight, hearing, smell, and taste was present on the anesthetic side of the face. The explanation of a case like this is perhaps not easily made, but the facts recorded with regard to the special senses are similar to those which are sometimes noted in hysterotraumatisms; and such a case serves to throw some light upon the apparent discrepancies of observation and the different inferences drawn with regard to the sense of taste after Gasserian and other operations. In some of the Gasserian ganglion cases previous operations have been performed on separate branches of the fifth nerve, and in these operations the chorda tympani may have been severed. In Finney's case, for example, in which taste was lost, the lingual had been divided in a previous operation, and it is not impossible that the chorda tympani at or after its junction with the lingual may have been severed in this and in other operations on the lingual at the angle of the jaw. Those cases which have been studied with most minuteness and scientific exactness by neurologists as well as by surgeons oppose the idea that the taste fibres are contained in any of the branches of the fifth nerve. In the second of the two cases reported by Finney, taste was not affected, although both the second and third branches of the nerve were cut and the ganglion was removed piecemeal. In four of the five cases recorded by Tiffany, in which taste was evidently carefully studied, it was entirely preserved, although the tongue was anesthetic on the operated side. Krause, in his monograph on Trigeminal Neuralgia, says that if his and other results are compared it will be seen that the statements regarding the preservation of taste prove more than do those which show loss or diminution of taste after operation on the Gasserian ganglion or on the nerves cephalad of this ganglion. He thinks, however, that it is necessary to give some satisfactory explanation of the cases in which taste has been disturbed after the operations. On the whole, his own results and those which he has collected are in favor of the view that the fifth nerve is not concerned with the gustatory function. The possibility of individual variations is suggested. In one case reported by Horsley, in which the nerve was twisted off at the spheno-maxillary fissure, taste, smell, and hearing were blunted on the affected side, which recalls the just cited observation of Bechterew.

The Bulbocerebral Gustatory Pathway and Cortical Centres.—The pathway from the terminal gustatory nucleus in the oblongata through the pons and midbrain to the cerebrum has not been clearly determined. The first neurons or nerve cells of the bulbocerebral tract originate from cells of the solitary bundle of the vagoglossopharyngeal nucleus. These soon pass to the raphe, decussating, according to Jakob, and uniting with either the lateral fillet tract or the superior median fillet tract. The continuation of the pathway is probably in the fillet tract to the cortex of the anterior inferior temporal convolution, probably the fourth temporal. Gowers records a case in which loss of taste was associated with loss of conjugate movement of the eyeballs to the same side, but without anesthesia in any of the areas supplied by the fifth nerve. This observation indicates the separation of the taste fibres from those of common sensation after entering the pons. Lesions of the posterior limb of the internal capsule have been associated with defect of taste on the opposite side, showing a decussation of the gustatory tract below this capsule. The cortical terminus of the gustatory apparatus, as shown under cerebral localization, is probably in the anterior inferior portion of the temporal lobe.

Clinical History.—*Special Gustatory Symptoms.* Affections of taste are analogous in mode of origin to those of smell. They may, for instance, be due to focal lesions of the gustatory apparatus, or they may be inflammatory or be primary degenerations involving the nerves of taste or their central tracts; or gustatory affections may be of hysterical and in rare instances of reflex origin. Like olfactory disorders, they may be unilateral or bilateral, and partial or complete. Whatever their mode of origin and distribution, they give certain more or less well defined symptoms. The patient may suffer from loss of taste (*ageusia* or *ageustia*), which may be unilateral, constituting *hemiageusia* (or *hemiageustia*); or his taste may be unusually acute, this symptom being known as *hypergeusia;* or, again, his taste may be perverted (*parageusia* or *parageusis*). Loss of taste is seldom absolute, but when it is the patient complains of everything being insipid, and often he becomes indifferent to food; at least he takes no special pleasure in partaking of it, and sometimes it becomes repugnant. Careful testing shows that the patient cannot appreciate any difference between substances which are sweet, sour, salt, or bitter. The taste for alkaline or metallic substances may also be abolished. In order to decide whether the loss of taste is complete, the sense should be tested not only on the tongue, but also on the lips, the inner surfaces of the mouth, the palate, the pharynx, and the epiglottis, and, if possible, even in the larynx. Often the patient is greatly disturbed by the loss of taste, and may become depressed and hysterical. The appetite frequently fails. Hemiageusia, or loss of taste confined to one side of the tongue, palate, and other surfaces

concerned with taste, is more frequent than the total abolition of this sense, but even hemiageusia of a complete type is not common. Most frequently the loss of taste is observed at the tip and lateral portion of the tongue on one side in the region of the gustatory fibres of the chorda tympani nerve. The cases in which the sense of taste has been abolished in this region are more numerous than those in which it has been impaired or abolished in the posterior half and in the palatine region. Occasionally taste is lost in islets of the mucous surfaces concerned with this function. When the loss of taste is local, and even in some cases when it extends over a large unilateral area, the patient may not at first complain of the defect, articles of food and drink being so rapidly distributed over all portions of the tongue and mouth that their taste is quickly appreciated by the unimpaired portion of the peripheral gustatory apparatus. Occasionally the patient and the physician first become aware of partial ageusia as the result of examination in cases where other symptoms attract the chief attention, as in paralysis of the seventh or facial nerve or of the motor and sensory subdivisions of the fifth nerve, or when paralysis affects any one or more of the cranial nerves from the fifth to the ninth. In some instances the ability to appreciate certain special qualities of taste is most affected: thus, the power to discriminate between sweetness and bitterness may be lost, while the patient may be able to distinguish sourness or saltiness. The normal taste becomes, of course, in a great degree developed and intensified in those whose occupations or inclinations lead them to the careful and skilful employment of this sense, as in wine tasters, tea tasters, and epicures. These unusual powers of discrimination by taste or by both taste and smell may be lost when the ability to distinguish between the standard qualities of taste is retained. In hypergeusia sapid substances are intensely and almost painfully appreciated, just as in some cases the organs of hearing are hypersensitive to sound and those of sight to light. The standards for taste vary somewhat, not only with education and training, but also with nationality and race. The insane frequently have hallucinations and illusions which take the form of gustatory sensations of an intense or acute type. Parageusia is sometimes an isolated symptom, but more frequently it occurs in connection with partial ageusia or hypergeusia. It shows itself as an alteration of the sense of taste amounting sometimes to an almost complete inversion, as when things which are sweet appear bitter or metallic. Substances which under normal conditions are agreeable may become disagreeable or loathsome, or the reverse of this may be observed. The patient may complain of all sorts of unpleasant gustatory sensations, which may be blended with perversion of common sensibility, as when the complaint is of an unpleasant taste with burning or tingling sensations in the tongue. One of the rather uncommon forms of epileptic aura is that in which the patient

complains of a peculiar taste which may or may not be associated with an odor. The taste may be offensive or pleasant, but it is more frequently the former. It may be regarded as either a hypergeusia or a parageusia, and is probably a sign either of local cerebral irritation or of disease of the olfactory or gustatory cerebral tracts or centres.

Secondary Sensations of Taste, or Taste Photisms. Secondary sensations of taste, or taste photisms, are rare as compared with light and sound photisms. In certain individuals agreeable and delicate tastes and smells call up agreeable and delicate shades of color, and disagreeable gustatory and olfactory sensations evoke correspondingly disagreeable colors. A taste photism is usually referred to that part of the mouth which receives the sensation. (Bleuler.)

Symptomatology of Focal Lesions of the Peripheral Gustatory Apparatus. Lesions in the gustatory apparatus anywhere from the taste buds to the cortical centres for taste may give rise to disorders of taste. In diseases affecting the mucous membrane of the tongue, the soft palate, and other peripheral gustatory regions, the taste buds may become involved, as in glossitis, or in catarrhal and in ulcerative affections of the mouth, just as anosmia may result from disease of the Schneiderian mucous membrane. The taste buds and the terminals of the glosso-pharyngeal and chorda tympani nerves are at times affected by toxic agents, or in rarer instances by injuries. Occasionally a unique peripheral gustatory affection is met with that is difficult of solution. Several years since, I saw a case in which an affection of both taste and common sensibility appeared to be due to the action of a vulcanite dental plate. This patient, whom I saw with Dr. O. B. Gross, of Camden, New Jersey, was a woman sixty-one years old, of nervous temperament. A short time after the vulcanite plate was put in her mouth she began to have an unpleasant taste, and soon she had also almost constant burning and tingling sensations of the tongue and lips. After about four months she ceased to wear the plate, but the burning sensation, which was confined to about one inch of the anterior extremity of the tongue on both sides, continued. Nothing seemed to taste to her as it should, and she complained of always having a coppery taste. Her mouth was dry, especially at night. Her lips and tongue were extremely sensitive to hot liquids and hard substances. The most reasonable explanation of such a case as this, if it was not hysterical, is that the influence of the plate or some of its ingredients upon the end organs of taste and of common sensibility in the tongue and lips caused a form of terminal neuritis with parageusia. When the loss of taste is due to lesions localized in the chorda tympani or pars intermedia of Wrisberg, it is, as before indicated, confined to the anterior and lateral portions of the tongue, and, as a rule, the ageusia is associated with other symptoms which indicate the position of the lesion. The affection is common, for

instance, in peripheral facial paralysis; and many cases have been reported in which it has been present when the lesion has been located anywhere in the course of the nerve from the entrance of the internal auditory meatus to the stylomastoid foramen. Very few intracranial peripheral cases of Bell's palsy with loss of taste have been recorded, but in such cases testing for taste is generally neglected. In one case recently studied by me of old facial and auditory paralysis evidently due to a syphilitic lesion at or near the so-called superficial origin of the facial and cochlear (auditory) nerves, taste in the peripheral distribution of the chorda tympani nerve was abolished. When the glossopharyngeal is affected anywhere in its course the loss of taste will be present on the posterior aspect of the tongue and in the palatal and other regions supplied by this nerve. (See Figs. 346 and 348.) In the rare case of a lesion invading the foramen or its immediate vicinity either just inside or just outside of the skull the symptoms show implication of the pneumogastric and spinal accessory nerves, the cervical sympathetic, the jugular vein, and the petrosal sinuses. While a considerable number of cases of aneurism, tumor, and other gross lesions involving the pneumogastric and spinal accessory in the neck have been recorded, the glossopharyngeal either has not been involved or observations demonstrating its implication have been overlooked. In such cases, besides the loss of taste in the posterior portion of the tongue and in the palatal regions, the symptoms to be expected would be such as more or less difficulty in swallowing, slow or irregular respiration and cardiac action, spasm or paresis of the muscles supplied by the spinal accessory nerve, laryngeal disorder, and symptoms of venous obstruction. When the lesion is in the intracranial course or at the superficial origin of the glossopharyngeal the symptoms will show involvement of the neighboring nerves and tracts according to the size and extensions of the lesion. In Pope's case (page 688), for instance, the patient, in addition to loss of taste, had diminution of power in the limbs, difficulty in swallowing, and other symptoms which showed involvement of the pneumogastric nerve and of some of the fibre tracts in the postoblongata.

Symptoms in Gustatory Affections of Nuclear Origin. In cranial bulbar paralysis of the nuclear type loss of taste is exceedingly rare. The nuclei usually involved are those for motor nerves, as that of the hypoglossal, the motor subdivision of the pneumogastric, the spinal accessory, the motor nucleus of the glossopharyngeal and of the facial. In acute apoplectiform bulbar paralysis the nucleus of the gustatory portion of the glossopharyngeal, including that for the pars intermedia of Wrisberg, may be involved; but reports of cases of this kind with loss of taste are rare. Tumor, softening, hemorrhage, or other gross focal lesion may involve the solitary fasciculus and cause disorders of taste.

Symptoms associated with Loss of Taste in Oblongatal and Pontile Lesions. When a lesion causing a disorder of taste is situated in the substance of the oblongata or the pons, at a point removed from the glossopharyngeal nucleus, the associated symptoms will vary according to the other nuclei and root fibres and tracts implicated in the lesion. In a case reported by Gowers, to which reference has been made on page 698, a tumor was supposed to be present in the pons near the level of the origin of the fifth. The patient had paralysis of conjugate lateral movements to the right, paralysis of the masticatory muscles of the same side, and entire loss of taste over the half of the tongue and the palate of the same side, but without any anesthesia. In a recent case of cerebrospinal syphilis studied by the author the patient had distinct loss of taste in the anterior portion of the tongue on one side, and some impairment of taste in other portions of the tongue. In this case, as in Gowers's, sensation was not impaired. Complete paralysis of the right external rectus muscles, paresis of the right side of the face, deafness on the left side, and some loss of hearing on the right, were present. Smell was also markedly affected, anosmia being complete on the left and almost complete on the right side. Sight was considerably impaired, the disks being pallid, but not atrophic, and the patient had polyuria. The lesion in this case was probably situated in the pons in such a position as to involve the root fibres of the abducent, and to a less degree those of the facial, auditory, and pars intermedia, or the bulbocerebral auditory and gustatory tracts in the pons may have been involved. In another case observed by the author, presumably of a syphilitic lesion at the base, taste was lost on both the anterior and posterior portions of the tongue; the other symptoms present being complete paralysis and anesthesia of all the muscles of the right side of the face, marked diminution of hearing on the same side, and a ringing sensation in the right ear. In this case a large gumma probably occupied the lateral aspect of the pons and oblongata. In a case which occurred in the Polyclinic service of the author, and which has been put on record by Drs. Bundy and McConnell, paralysis of the right external rectus, slight drooping of the right side of the face, abolition of pain sense on the right side of the head and face (tactile and temperature sense being unimpaired or but slightly affected), almost complete deafness in the right ear, complete loss of taste on the right side both anteriorly and posteriorly, and paralysis of the left arm and leg, were present. The authors in reporting this case are inclined to attribute the complete loss of the sense of taste on the right side to a lesion affecting taste by way of the trigeminal nerve, believing that the absence of paralysis of any of the muscles supplied by the glossopharyngeal nerve gives a valid objection to the idea that this nerve or its root fibres were damaged. Instead of this explanation it seems, however, more reasonable to

believe that the gustatory pathway from the glossopharyngeal nuclei through the pons to the midbrain and higher regions was implicated in the lesion. Although the course of this tract, as has been stated, has not yet been clearly determined, it is probable that it is in the lateral or superior mesal fillet.

Symptoms in Lesions of the Cortical Gustatory Area. As stated on page 348, the cortical area for the sense of taste has not yet been positively determined. but it is probably in the inferior aspect of the temporal lobe, and in part at least in the fourth temporal convolution. Anderson has recorded a case of cerebral tumor affecting the left temporal lobe at the base. It arose from the pituitary body, occupied the space of the intrapeduncular ganglion, and grew chiefly to the left and backward, so that the first part of the cortex involved by it had been the anterior part of the inner border of the left temporal lobe. The patient had epileptiform attacks preceded by a rough, bitter sensation in his mouth, which remained during the attack. Smell was normal in the right nostril, but defective or absent in the left. Taste was apparently defective on both sides. Other phenomena were present to which reference need not here be made. In cortical tumors in the gustatory areas we should therefore expect to have auras of a gustatory type, and later more or less complete loss of taste.

Loss of Taste from Encephalic Lesions variously situated. In several cases of cerebral tumor loss of taste has been among the symptoms present. Thus, in a case reported by Howe, the different symptoms were loss of sight, increasing to total blindness, and gradually increasing loss of hearing, of smell, and of taste, in the order named, no anesthesia nor paralysis being mentioned. The autopsy showed a fibrosarcoma involving the inferior portion of the right anterior (prefrontal) lobe. The first and second pairs of nerves were involved, but no others. In another case, recorded by Broadbent, the symptoms were mental depression, right-sided temporal and orbital neuralgia, anesthesia of the left arm, slight attacks of spasm with unconsciousness in the left face, left arm, and left hand, paresis of the left arm, blindness, optic neuritis, dilated and immovable pupils, and loss of smell and taste. The autopsy showed gummata in the right supramarginal lobule, two tumors the size of a pea lying superficially. Eskridge has reported a case of tumor of the right lateral lobe of the cerebellum, with loss of taste on the side of the tumor, associated with other typical symptoms, local and general, of an encephalic neoplasm; and Formad has reported another case, a tumor of the left lateral lobe of the cerebellum and the vermis, in which during the last days of life the patient suffered from loss of power of deglutition and also from loss of taste, of smell, and of hearing.

Gustatory Affections in Tabetic Cases. In rare cases of posterior sclerosis and of other chronic degenerative neuraxial diseases I have

observed loss of taste in the region supplied by the gustatory subdivision of the glossopharyngeal. In some of these cases it is probable that the petrous and jugular ganglia become affected with degenerative disease similar to that which attacks the spinal ganglia. Althaus has recorded a case of tabes in which the patient had lost the power of distinguishing flavors, smell and taste both being apparently affected. Both hyperosmia and hypergeusia, at least hypersensitiveness of taste, are sometimes observed in tabes, and in these cases the odors are usually offensive and everything that is eaten has an unpleasant flavor. The gustatory impressions are never of an agreeable character. They are not infrequently premonitions of mental derangement such as occurs in the tabetic.

Affections of Taste of Hysterical Origin. Affections of taste are rarely of hysterical origin, although cases are occasionally seen which will hardly bear any other explanation. Although I have tested for taste in a number of cases of hysterical hemianesthesia and hemiparesis with other stigmata, I have not yet observed a case in which taste was impaired or absent. Recently I examined a case of almost universal anesthesia, only islets of retained sensation being here and there present, but taste was fully preserved. Hysterical, or at least neurotic, individuals often seem to have an unusual acuteness of taste, detecting ingredients in food or medicine which may not be appreciable to others. In consultation with Dr. Samuel Ayres, of Pittsburg, I saw an interesting case of bilateral loss of taste, insalivation, partial analgesia over the anterior two thirds of the tongue and inner surfaces of the mouth, and diminished tactile and pain sense in the region supplied by the great auricular and small occipital nerves. The patient was a woman of nervous temperament, and had undergone much strain and worriment. Her appetite failed, and she became indifferent as to what she ate or whether she ate at all. She first noticed that sweet substances, such as sugar, did not taste so sweet as they should, and she soon observed that all vegetables were insipid. Everything had a sort of metallic taste, and eventually all food became repugnant. Sleep became irregular and unrefreshing, and she frequently felt despondent, with a sense of impending trouble. The patient was undoubtedly neurasthenic. She eventually recovered. The disorder of taste was probably, but not certainly, of hysterical origin.

Etiology and Pathological Anatomy.—The etiology of disorders of taste varies according to the nature of the lesion causing them. The majority of cases is due to focal lesions. A large number of cases is found in association with peripheral facial paralysis (see article on facial paralysis). Other recorded focal causes, which have been more fully referred to in discussing the gustatory path and centres and the symptomatology of focal lesions, are exostosis, purulent middle ear disease with caries, injury to the lingual nerve, neu-

ritis, basal fracture, tumor, softening or other lesion of the pons or oblongata, nuclear degeneration, and tumor or other focal lesion of the anterior inferior portion of the temporal lobe. Substances introduced into the mouth may act as causes, as in the case seen with Dr. Gross, in which the apparent causative agent was a vulcanite dental plate. The use of extremely cold or hot substances, of acids, chewing gum, tobacco, or the application of any other substances which overstimulate the gustatory apparatus, may lead to impairment of taste. The same effect may be brought about by catarrhal or other diseases of the mucous membrane of the tongue, palate, and palatine regions. Meningitis of syphilitic or tubercular origin may give rise to affections of taste. Nixon has put on record a case in which the patient suffered from double facial paralysis, deafness, and other symptoms, including bilateral loss of taste. Nixon attributed the symptoms to localized meningitis of syphilitic origin. The disease probably extended from the surface of the temporal lobe along the Fallopian canal, and from there to the tympanic cavities. The morbid appearances in affections of taste of organic origin are those of neuritis or perineuritis of the nerves affected, of tumor, hemorrhage, softening, or other focal lesion of the gustatory pathway, and in some instances of nuclear, root fibre, or tract degeneration. The most interesting matter in connection with the pathology of the sense of taste has reference to the exact location of the lesion producing it, and this has been fully considered under focal lesions.

Diagnosis.—In the first place, a critical examination to determine the presence or absence of the sense of taste should be made,—a fact important to remember, as affections of smell and of common sensibility are sometimes supposed by the patient and even by the physician to be disorders of taste. The diagnosis of the nature and location of the lesion has already been sufficiently discussed. Hallucinations and illusions of taste have an unusual diagnostic importance, because they frequently lead their victims to believe that they are the subjects of conspiracy or persecution, or even of attempts upon their lives, by poisoning. Such patients transform sensations of taste which are the result of indigestion, or are purely mental, or are due to the condition of the secretions of the mouth, into evidences that their food and drink are being tampered with, or they believe medicines are given to them for the sake of doing them injury. It is a not infrequent experience for well known chemists to have samples of food or drink brought to them for analysis by those who are sufferers from this form of delusion. The diagnosis in such cases has to be made by a careful study of the history and mental condition of the patient. Usually such hallucinations are associated with other evidences of delusional insanity. The modes for testing for taste have been already discussed in Chapter II., page 165. As there stated, the distinction between smell and taste and between taste and

touch must always be carefully made. Care should be taken to test all the portions of the tongue, mouth, and pharynx which are supposed to be concerned in taste. Each half of the tongue and each peripheral gustatory area should be examined separately. A sapid substance can be applied by means of a glass rod or brush after the methods indicated on page 165. A patient's statement with regard to taste should not be depended upon without examination, as sensations due to involvement of the nerve of common sensibility may be attributed to loss or perversion of taste, or may be so regarded by the patient.

Prognosis.—The prognosis varies with the character and position of the lesion. Hysterical cases have the same prognosis as hysteria in general. Cases due to peripheral neuritis or other peripheral lesion are sometimes amenable to local medicinal, electric, or surgical treatment. As the greater number of central cases is due to destructive or degenerative lesions, their prognosis is generally bad.

Treatment.—Treatment of affections of taste must be based upon clear views as to their nature. When loss or perversion of taste is attributable to a tumor situated in the temporal lobe or anywhere in the corticobulbar tract, the treatment would be such as is indicated for brain tumors in general. In like manner, if the lesion causing the disorder of taste is a hemorrhage, area of softening, abscess, or other destructive lesion, no treatment except that applicable to the lesion would be of any avail. The mistake is sometimes made of using too active peripheral treatment in cases of impaired taste due to central lesion. If the loss of taste is due to a neuritis involving the chorda tympani or the lingual or glossopharyngeal nerve, the treatment is that indicated for localized neuritis wherever situated. In syphilitic cases antisyphilitic remedies should be employed. Efforts are usually made to improve or cure affections of taste by local applications of the faradic or galvanic current, or by the use of stimulating substances to the tongue and mucous membrane of the palate. These methods can be of service only when reason exists for believing the gustatory disorder to be either the remains of a peripheral nerve lesion or of hysterical origin. In a few cases surgical treatment has been resorted to, as in Gross's case referred to on page 700, in which, after various remedies, local and internal, had been unsuccessfully tried, section of the lingual nerve on one side was performed. The patient made very considerable improvement, but did not entirely recover. The disease was bilateral in its manifestations, but the pain and discomfort suffered by the patient were most persistent and severe on the right side. Dr. Gross removed the painful area of the mucous membrane of the tongue, and cut away the tissues in the submaxillary gland, his object being to dissect away those portions of the nerve supplying the right edge of the tongue which were believed to be locally diseased.

AFFECTIONS OF HEARING DUE TO DISEASE OF THE COCHLEAR NERVE AND ITS RELATED ENCEPHALIC STRUCTURES.

Subdivision of the Eighth Nerve.—While for several years the distinction between the true auditory or cochlear nerve and the vestibular nerve or nerve of equilibration has been clearly recognized, no efforts have been made by clinical writers to separate the affections of the one nerve from those of the other. The separate courses of these two nerves have been traced from their origins to or nearly to the midbrain. Their diseases will be considered separately, although the clinical data for the vestibular nerve are meagre. Peripheral, oblongatal, and pontile affections of the two nerves may be associated in the same case, as when a lesion involves the petrous bone, when diffuse disease of the labyrinth is present, or when focal lesions of the floor of the skull or of the oblongata or pons involve various structures. Cases are met with in which the symptoms point clearly to disease limited to the peripheral portion of either the cochlear or the vestibular nerve. The origin and course of each of these nerves have already been briefly considered in Chapter I. (page 81). In the table on page 105, showing the constitution of the different sensory tracts and their homologues, the origin, course, and termination of the cochlear nerve are given. Some differences of opinion exist as to the methods of peripheral subdivision of the cochlear and vestibular nerves. Retzius classes the ramus medius of Schwalbe, or sacculo-ampullary nerve, with the cochlear nerve, while others regard it as a subdivision of the vestibular nerve, or at least as being more closely allied to the latter than to the former. Retzius subdivides the eighth or acoustic nerve into an anterior and a posterior branch. The anterior branch (the superior branch of Schwalbe), known also as the utriculo-ampullary branch, is the vestibular nerve as generally recognized. Its three branches come through the superior cribriform spot (*macula cribrosa superior*) from the macula acustica of the utricle, the crista acustica of the anterior ampulla, and the crista acustica of the external ampulla. The ramus medius of Schwalbe, whether classed with the vestibular or with the cochlear nerve, is apparently a subdivision of the posterior trunk. One of its two branches passes through the middle cribriform spot (*macula cribrosa media*) from the macula acustica of the saccule; the other through the inferior cribriform spot (*macula cribrosa inferior*) from the crista acustica of the posterior ampulla. The ramus inferior of Schwalbe, or cochlear nerve, comes from the spiral ganglion through the central foramen of the cochlea.

The Essential Nerve of Hearing.—The cochlear nerve is the essential nerve of hearing,—the nerve which is absolutely necessary to the function of audition. Formerly by some high authorities the

cochlea and semicircular canals were both supposed to play important rôles in the sense of hearing. The cochlea, for instance, was thought by Helmholtz to be concerned especially in the analysis of sounds; and different portions of its nervous apparatus have been assigned special functions. Complete but isolated destruction of the cochlea in dogs has, however, resulted in total deafness. The secondary connections of the auditory nerve are exceedingly complex, because not only do sensations from the ear play a most important part in orientation, "but auditory impulses start innumerable protective movements which also take part in the highest degree in intellectual life." (Hill.) It remains true, nevertheless, that the cochlear nerve is the essential nerve of hearing.

Accessory Nerves of Hearing.—Audition is provided with an accessory apparatus in some respects more important than that for any of the other special senses, and the nerves of this apparatus may be regarded as the accessory nerves of hearing. Some of these nerves are concerned with the regulation of muscular movements which render audition more facile; others are nerves of common sensibility to the membrane of the tympanum and to other parts of the auditory apparatus; and while the vestibular nerve is now regarded as the handmaid of a separate and special sense, that of equilibration, it probably plays some important accessory rôle in the auditory function. Gruber and some others still believe that the semicircular canals and their nerves take some part in audition. The delicate posings of the head and of the body often resorted to for the better appreciation not only of the direction of sound but also of variations of tone and pitch point to one of the probable uses of the vestibular nerve in connection with hearing. The tympanic membrane first informs as to whether the sound is from within the body or from without; and the power of making this distinction depends on the tactile perception of the membrane. Hearing, therefore, is blunted but not lost in some hysterical and in some organic hemianesthesias. The tactile perception of the external auditory canal may also play a minor part in acute hearing. Diverse movements accompany the processes of audition; and, broadly regarded, all the nerves and muscles concerned in the placing of the head, body, and eyes in different positions in order to appreciate sounds are portions of the accessory apparatus of hearing, but the tensor tympani and the stapedius muscle and their motor nerves are in an especial sense portions of this accessory apparatus. The nerve to the tensor tympani is derived from the otic ganglion, and is a branch of the fifth; that to the stapedius is a filament given off from the facial in the Fallopian aqueduct. Contraction of the tensor tympani muscle increases the labyrinthine pressure by augmenting the tension of the tympanum, while contraction of the stapedius muscle diminishes its tension and decreases labyrinthine pressure.

These movements assist in the prevention of injurious effects from loud sounds. It is sometimes particularly important when hearing is diminished, but not lost, to determine whether this impairment is due to the implication of the accessory apparatus, as in facial paralysis, in some affections of the fifth nerve, and in some cases of hemiplegia or hemianesthesia.

Peripheral Auditory End Organs.—In the membranous portion of the cochlea are the terminations of the cochlear or true auditory nerve. The extreme peripheral portion of the auditory apparatus is composed of what are known as *hair cells*, which are in reality modified epithelial cells, homologous with the taste buds and with the tactile end apparatus. They are not nerve cells, but are in contact with the processes of nerve cells. They are simply columnar epithelial cells, and are subdivided into an *inner* and an *outer* set, which are provided with numerous hairlets near their ends. Beneath the hair cells are found the sustentacular cells of Deiters. The ends of Deiters's cells separate the hair cells and unite, forming a cuticular network, through the openings of which the hair cells pass to the surface. Within, the inner row of the cells of Deiters rest upon the rods of Corti. The inner hair cells have no connection with Deiters's cells, but rest on the inner rods of Corti. The pillars of the rods of Corti consist of rows of columnar epithelial cells, which, coming together, form an arch; they are more than three thousand in average number. The whole epithelial and sustentacular structure is covered by a fibrillated membrane known as the tectorial membrane. The term *organ of Corti* is applied to this entire specialized epithelial structure.* The true nervous structure of the auditory apparatus begins with the terminal ramifications of the dendritic processes of the cells of the spiral ganglion, the other processes passing centrally into the true cochlear nerve. The cells of the spiral ganglion are bipolar. A small canal winds around the modiolus, the central axis of the cochlea, and in this canal numerous ganglion cells constitute the spiral ganglion. Retzius first presented the view of close contact of nerve terminals and epithelial acoustic

* The name of Corti is applied to several important labyrinthine structures, and should be used correctly. As here stated, the term organ of Corti is usually applied to the entire epithelial structure which constitutes the end organ of the auditory apparatus. Sometimes it is incorrectly used to designate the epithelial arches or inner portion of this apparatus. The ganglion of Corti is a part of the true nervous apparatus, but the term ganglion of Corti is sometimes used in two different ways: *habenula ganglionaris of Corti*, for example, is the name applied by some to the entire ribbon-like spiral ganglion which is the true origin of the cochlear nerve; and the branch of the inferior portion of the auditory nerve, which has already been described as the ramus media of Schwalbe, has upon one of its branchlets which cross to the ampulla of the posterior semicircular canal a small ganglion, also sometimes called the ganglion of Corti.

cells, showing that one was not a direct continuation of the other, this being the first intimation that the organ of Corti was not a true nerve structure. The acoustic cells are therefore, as shown by Lenhossék, different from the olfactory and retinal cells, at least so far as our present knowledge is concerned. Since Retzius, the fact of the free termination of the dendritic fibrils of the spiral ganglion has been demonstrated by Ramón y Cajal and other observers. It has been denied by some, but not on exact histological evidence.

Course and Terminal Nuclei of the Cochlear Nerve.—The so-called eighth nerve consists, as already stated, of two strong root

FIG. 349.

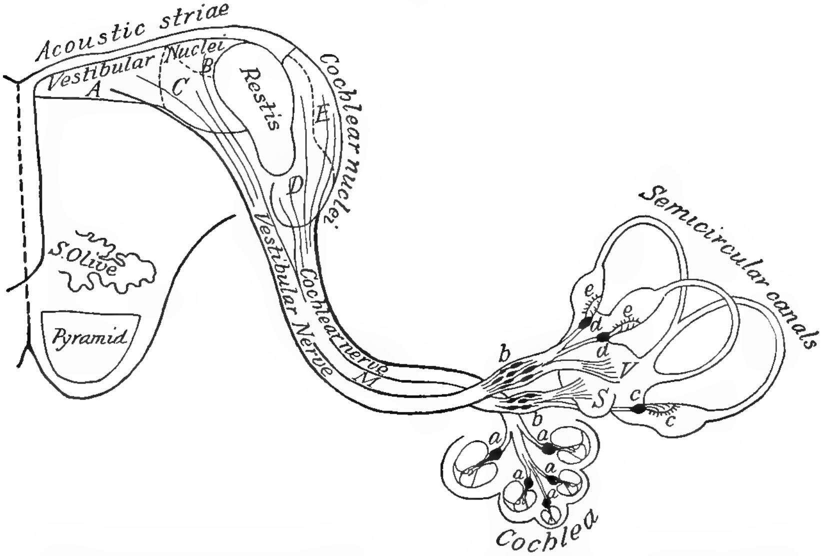


Diagram of the cochlear and vestibular nerves, showing their origin, peripheral expansions, trunks, and terminal nuclei in the bulb: *a, a, a, a, a*, the spiral ganglion as shown in a section of the cochlea; *b, b*, Scarpa's ganglion; *c*, Corti's ganglion in the ampullæ of the inferior or posterior semicircular canals; *d, d*, unnamed ganglia in the ampullæ of the superior vertical or anterior and in the horizontal or transverse semicircular canal; *e, e, e*, cristæ acusticæ; *V*, utricle; *S*, saccule; *M*, ramus medius of Schwalbe; *A*, dorsomesal nucleus; *B*, Bechterew's nucleus; *C*, Deiters's nucleus; *D*, ventrolateral nucleus; *E*, acoustic tubercle.

bundles, which become the cochlear and vestibular nerves. In the internal auditory meatus the seventh and eighth nerves and the pars intermedia of Wrisberg lie together, the eighth being in the lowest plane. Here the vestibular and cochlear nerves are united in the same bundle and together pass towards the cerebrospinal axis, between the medipeduncle and the pneumogastric lobe of the cerebellum to the most cephalic portion of the postoblongata, which they enter at the location usually described by gross anatomists as the superficial origin of the eighth nerve. Just as these nerves have originated from separate ganglia, so they now again separate as

they enter the postoblongata into two systems of roots, an *external*, or cochlear, and an *internal*, or vestibular. The cochlear root passes around the postpeduncle, and its constituent fibres reach the *ventrolateral nucleus*.*

Distribution and Course of the Cochlear Nerve in the Brain Stem.—The fibres of the cochlear nerve, after entering the ventrolateral nucleus, divide into an ascending and a descending root, each having numerous collaterals. According to Held, some branches of the fibres of the cochlear root form around the cells basket-shaped masses similar to the Purkinje cells. Ramón y Cajal has reinvestigated the course of the cochlear fibres to the brain stem, his studies having been made on the brains of newborn mice and rabbits. According to him, the ascending branch passes cephalad and dorsad, and terminates at the level of the ventral nucleus, after giving origin to numerous collaterals. This branch in its course enters the tail of the nucleus and the acoustic tubercle. Axis cylinders from all parts of the nucleus pass cephalad into the trapezoid body. Numerous terminal fibres are found in the ventral nucleus, which appear to have come by way of the trapezoid body from the ventral nucleus of the opposite side. While most of the large number of fibres which constitute the external root terminate in the ventral nucleus, a portion of them passes through this nucleus, and cephalad through the superior olives, the trapezoid nucleus, and the nucleus of the lateral fillet, to the postgeminum; while still another portion, after passing through all the structures just named, continues onward, and probably reaches the cortex of the opposite temporal lobe. Held and Ramón y Cajal differ somewhat with regard to the relations of the superior olive to the acoustic pathway. According to Held, the axis cylinders of the bipolar cells pass not only to the ventral nucleus, but partly to the trapezoid body and partly to the peduncle of the superior olive, the latter terminating in the nucleus of the abducent nerve. Ramón y Cajal was able in only one case to see the axis cylinders directed to the nucleus of the abducens, and he was not able to discover its

* This ventrolateral nucleus consists of two parts, each of which has a variety of names. The more ventral is perhaps more commonly spoken of as the *ventral nucleus* (*accessory nucleus, lateral acoustic nucleus, lateral nucleus of the anterior root, anterior nucleus, ventral accessory acoustic nucleus, auditory ganglion*); the more dorsal and lateral is generally designated as the *acoustic tubercle*, but is sometimes known as the *tuberculum laterale*, or lateral tubercle, and as the *superficial auditory nucleus*. The terminology employed for the gross and microscopic structures of the pons and oblongata is often of a confusing character. The genius of a Wilder is needed to rid us of the medley of terms used to describe the same structures. If a single term, and that a mononym, could be chosen for each of these structures and adhered to by anatomical and neurological writers, it would be of great advantage. To make the confusion greater, different and equally distinguished authorities sometimes use one term to designate different structures.

entrance into the cells of this nucleus.* By the name *nucleus preolivaris* Ramón y Cajal designates a considerable mass of cells anterior to the superior olive and external to the trapezoid nucleus. He traced from this body a central tract which was not observed by either Held or Koelliker, believing that it corresponds to the bundle which Held considers as being in union with the nucleus of the abducens. He holds that he has discovered an entirely new nucleus connected with the acoustic apparatus, which he designates as the *nucleus semilunaris* or *preolivaris externus* because it is situated around and superior to the convexity of the superior olive. This nucleus is preeminently characterized by the presence, surrounding it, of one, two, or more bundles of collaterals of such extraordinary delicacy that they represent without doubt the finest fibrils in the central nervous system. The nerve processes of the cells of the trapezoid nucleus, according to Held and Koelliker, pass in the trapezoid body towards the olive and the raphe, sending off collaterals. Ramón y Cajal, whose description of the course and termination of the processes of the cells of the trapezoid nucleus differs in no essential particulars from that of Held, found that the fibres which end in the trapezoid nucleus are of three kinds: collaterals of the trapezoid body, ramified terminal fibres, and terminal fibre baskets or fibres of Held. The trapezoid body, in a restricted sense, represents a central tract of the terminal acoustic nuclei, and also a transverse commissure between the cells of these nuclei. Ramón y Cajal found this formation highly developed in mice. The lateral lemniscus consists of fibres separated by masses of cells which can be divided into two special aggregations, known respectively as the *lower* and the *superior nucleus of the lateral lemniscus.* Koelliker and Held regarded the collection of cells which Ramón y Cajal designates as the lower nucleus of the lateral lemniscus as a continuation of the superior olive; but the latter believes that the cells of this nucleus are not similar to those of the superior olive. He found that their processes turn towards the raphe and do not ascend as represented by Held.

Diagrams of the Central Auditory (Cochlear) Tract.—In the three diagrams Figs. 350, 351, and 352 are shown the chief constituents of the central cochlear tract according to Held. In Fig. 350 is represented the direct or root fibre system of this nerve. The processes which form the cochlear nerve come, as already stated, from the ganglion spirale. One set of root fibres passes into the ventral nucleus, from the mesal side of which transverse fibres cross to the opposite side in the trapezoid body, which has relations to the superior olive of both sides. This trapezoid body contains gan-

* In Bruce's diagram, Fig. 242, page 380, the usually accepted connection of the nucleus of the sixth with that of the eighth nerve is shown.

glion cells of its own, constituting the trapezoid nucleus. Other cochlear fibres end in the acoustic tubercle, and connections are made between it and root fibres of the ventral nucleus which pass through the superior olives of both sides and reach the fillet nucleus of the opposite side. Still another set of root fibres passes through the ventral nucleus, and is connected by terminal branches or trees with the superior olives and trapezoid nuclei of both the same and the opposite side, continues on through the fillet to the lateral fillet nucleus, the postgeminum, and the pregeminum, and eventually

FIG. 350.

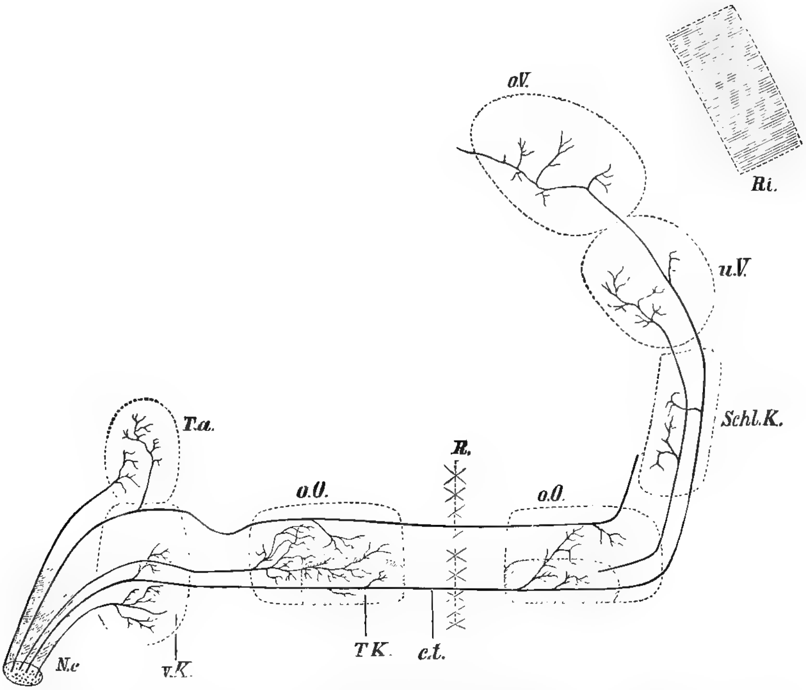


Diagram of the central auditory tract (direct or root fibre system): *N.c.*, cochlear nerve; *T.a.*, acoustic tubercle; *v.K.*, ventral nucleus; *T.K.*, trapezoid nucleus; *c.t.*, trapezoid body; *o.O.*, *o.O.*, superior olives; *R.*, raphe; *Schl.K.*, nucleus of the lemniscus; *o.V.*, pregeminum; *u.V.*, postgeminum; *Ri.*, cortex. (Rauber, after Held.)

reaches the cortex of the opposite temporal lobe. The figure just described gives only the "neuron" or nerve cell system of the first order. A root fibre system of the second order is connected with this first system, and is shown in Fig. 351. The fibres of this system arise from cells which lie within the ventral nucleus, in the acoustic tubercle, in the superior olives, in the trapezoid nucleus, in the nucleus of the lateral lemniscus, and in the quadrigeminal bodies. The acoustic striæ, or striæ medullares, run from the acoustic tubercle to the postgeminum of the opposite side. These striæ

contain also fibres which arise from higher centres and terminate in the acoustic tubercle, these belonging to the recurrent system. The fibres of this recurrent system (shown in Fig. 352) terminate in the ventral nucleus. They originate in the pregeminum and the postgeminum, in the lateral fillet nucleus, in the superior olive, and in the trapezoid nucleus of both the same and the opposite side. In addition to the systems illustrated in these three diagrams, another system of reflectory or reflex tracts exists, tracts which unite the auditory systems with other parts of the brain, chiefly motor in

FIG. 351.

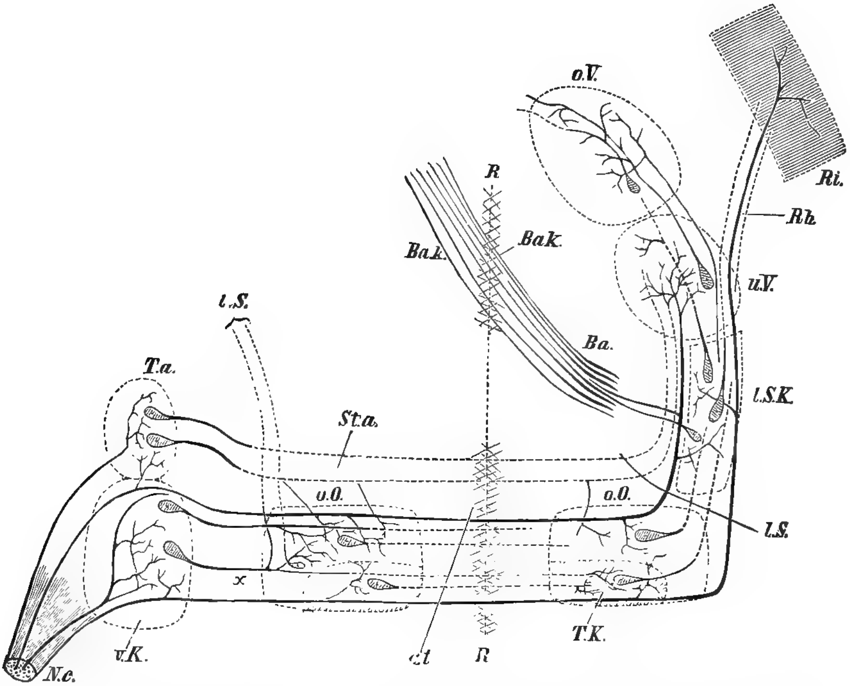


Diagram of the central auditory tract (system of the second order): *N.c.*, cochlear nerve; *v.K.*, ventral nucleus; *c.t.*, trapezoid body; *R.*, raphe; *T.K.*, trapezoid nucleus; *o.O.*, superior olive; *T.a.*, acoustic tubercle; *St.a.*, acoustic striæ; *l.S.*, lateral lemniscus; *l.S.K.*, nucleus of the lateral lemniscus; *Ba.*, prepeduncle; *Bak.*, decussation of prepeduncle; *o.V.*, pregeminum; *u.V.*, postgeminum; *Ri.*, cortex; *Rb.*, cortical tract. (Rauber, after Held.)

function. This reflex and associating system may be said to arise in the postgeminum, and has within its scope the optic nerve, the nuclei and root fibres of the abducens, facial, third, and fourth nerves, the dorsal longitudinal bundle, and the first cervical nerve. As is shown by these diagrams, the central auditory tract terminates in large part in the quadrigeminal body, both as regards its systems of the first and the second order, and here also originate its recurrent and reflex systems. According to some authorities (as shown in the table on

page 105), the postgeniculum constitutes a portion of the cochlear auditory tract. In the midbrain, therefore, the auditory tract divides into two great parts, one of which goes to the cerebrum to transmit auditory impressions to consciousness, the other sending out radiations concerned in the transmission of impulses which control the

FIG. 352.

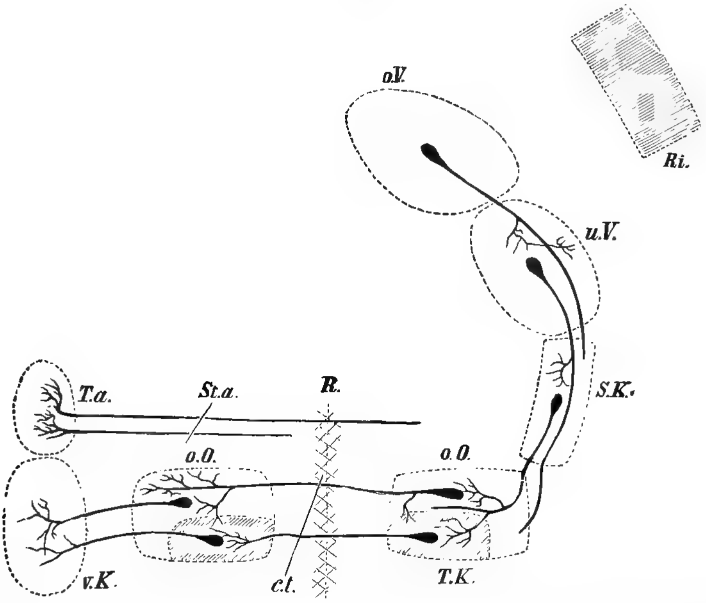


Diagram of the central auditory tract (recurrent system): *v.K.*, ventral nucleus; *c.t.*, trapezoid body; *R.*, raphe; *T.K.*, trapezoid nucleus; *o.O.*, superior olive; *T.a.*, acoustic tubercle; *St.a.*, acoustic striæ; *S.K.*, nucleus of the lemniscus; *o.V.*, pregeminum; *u.V.*, postgeminum; *Ri.*, cortex. (Rauber, after Held.)

numerous reflexes associated with audition. From the postgeminum and postgeniculum the central acoustic tract passes through the subthalamic region, thence probably by way of the posterior limb of the internal capsule (posterior to the sensory projection fibres) to the temporal lobe, which, as shown on pages 344 and 345, is the cortical terminus of the auditory pathway.

Clinical History.—*Definitions.* Loss of hearing, or deafness, is known as *anacusia* (*anacusis, anacousia, anacousis*). It may be unilateral (*hemianacusia*). Hyperacuity of hearing is *hyperacusia* (*hyperacusis, hyperacousia, hyperacousis*). Special terms are sometimes applied to particular varieties of hyperacuity of hearing. *Oxyacoa* or *oxyakoia* indicates the acoustic state in which an increased capacity for the detection of sounds of very low pitch is present. Acoustic hyperesthesia, or *dysacusia*, is an unpleasant or painful sensation in the ear caused by a noise or tone. *Paracusia acris* sometimes signifies an intense and incessant acuity of hearing. The general term *para-*

cusia, meaning perversion of hearing, is the analogue of parosmia and parageusia. It has many varieties, which are more properly discussed in works on otology. Thus, in *paracusia duplicata*, or *diplacusis*, sounds are heard double. *Paracusis loci* (*paracusis localis*), observed especially in unilateral deafness or when the two ears hear unequally, is a term employed to designate the difficulty of estimating the direction from which sound comes. *Paracusia Willisii*, or *paradoxic deafness*, is the form of impairment of hearing, somewhat frequently met with, in which the patient is able to hear better in the presence of a noise.

Special Symptoms of Lesions and Disturbances of the Auditory Apparatus. Diseases of the cochlear nerve and auditory apparatus are organic and functional, and these may be focal or diffuse, intrinsic or extrinsic, inflammatory or degenerative. Functional affections usually involve the entire auditory apparatus, as when a patient suffers from a neurasthenic or hysterical affection of hearing. Focal lesions are most frequently extrinsic,—lesions which cause irritation, destruction, or compression of the auditory structures. Inflammatory lesions are commonly diffuse, and in the majority of cases are conjointly intrinsic and extrinsic, implicating both the acoustic structures and their environment: perineuritis and neuritis, for instance, are usually combined in the same case. The symptoms may be referable to the auditory end organs; to the trunk of the cochlear nerve; to the ventrolateral nucleus of the postoblongata; to the auditory pathway from this nucleus to the cerebrum; and finally to the cerebral tracts and cortical centres for hearing. Separate and recognizable syndromes are also given by special lesions of the centres intercalated in the auditory pathway. Degenerative disease of the auditory apparatus presents itself most frequently as one of the evidences of degenerative disease of the cerebrospinal axis, as when tabetic atrophy is present in locomotor ataxia. In affections of the auditory apparatus two symptoms, partial or complete *deafness* and *tinnitus*, are of dominating importance, and most likely to persist or recur. Vertigo or dizziness, which is usually included among auditory nerve symptoms, is in reality a symptom referable to disease of the vestibular nerve or its related central parts. The cochlear nerve may be affected in any part of its course or through its entire length without any subjective or objective disturbances of equilibration, although more commonly some vertigo is present with the deafness and tinnitus, because in the majority of cases the cochlear and vestibular nerves are implicated together, particularly in peripheral affections. First brief consideration will be given to tinnitus and a few special auditory symptoms. Tinnitus is the name applied to the peculiar subjective noises heard in many forms of aural or auditory disease, and it is described by patients by such expressions as buzzing, hissing, blow-

ing, tinkling, ringing, whistling, and the sounds of bells or music. Speaking generally, tinnitus is a symptom of irritation, and when it is due to a lesion of the auditory nerve this is most frequently located in the cochlear portion of the labyrinth ; but many of its varieties are due to affections of parts of the auditory apparatus other than the nerve, as the external or the middle ear. It may be present even when all parts of the auditory apparatus are intact, as when it is psychical or of reflex origin. *Sound photisms* are sensations of light or of colors which result from the hearing of certain sounds. When these photisms are developed in the highest degree, their subjects have with every sound some sensation of color. A sound photism usually seems to proceed directly from the source of sound, and endures only as long as the sound. The various forms of hyperacuity of hearing need brief special discussion. In true hyperacusia the power of appreciating sounds is increased, so that sounds which are not heard by others may be heard, or ordinary sounds may be perceived with augmented and perhaps even painful intensity. A true hyperacusia is rare, but is sometimes observed in hysterical subjects and in connection with irritative disease of the auditory apparatus. Auditory hyperalgesia is more common than true auditory hyperesthesia. In this condition sound itself is not heard with increased intensity, but painful sensations accompany the sounds which are heard. It is sometimes spoken of as dysesthesia or dysacusia. The patient suffering from cerebral meningitis, neoplasms, abscess, or other irritative intracranial lesions may exhibit this symptom in the highest degree, the slightest sound causing pain and great nervousness. Various forms of paracusia and hyperacusia may be due to the same or to similar causes. In rare instances a form of auditory allochiria is observed, sounds made on one side of the head or neck being heard only on the opposite side. Auditory hallucinations are of common occurrence in several varieties of insanity, but the proper place for their full discussion is under mental disorders. Hallucinations and illusions of hearing are, however, dependent at times upon lesions or functional disturbances of the auditory apparatus, and may occur among the sane. Hallucinations due to cortical irritation or exhaustion may be increased in frequency and intensity by abnormal states of the peripheral apparatus for hearing. Purely imaginary or hallucinatory sensations of hearing are to be distinguished from *entotic noises*, which can be referred to some abnormality within the organ of hearing, and from *periotic noises*, due to lesions or disturbances near but not in the ear. Among the sources of entotic noises are changes in the circulation of the bloodvessels of the internal ear, vibration of the ossicles, and contractions of the tensor tympani or the stapedius muscle. In rare cases, in anemic patients, sounds due to the movement of blood in the carotid artery are heard, and in other rare instances

the sounds heard may be due to an intra-aural or an intracranial aneurism. Such sounds are more acutely perceived when the nerve is in a hypersensitive state. Noises in the ear complained of by the patients can rarely be heard by others; but an exception to this is when a crackling noise, the result of contraction of the muscles of the Eustachian tube, is heard in the maxillary articulations.

Symptoms of Focal Lesions of the Auditory Apparatus. Whether primarily or secondarily involved, the epithelial end organs and the spiral ganglion and its processes in the cochlea are the portions of the peripheral apparatus for hearing most frequently affected by organic disease. The chief symptoms of lesions of these structures are deafness and tinnitus; if partial at first, the deafness usually sooner or later becomes complete, as the lesions are commonly either of a destructive or of a degenerative character. Testing for hearing with the watch, the tuning fork, and other appliances shows impaired response both by bone conduction and by aerial vibration. A gross affection involving the trunk of the cochlear nerve almost necessarily invades the vestibular nerve trunk, because of the close association of the two nerves in their course from the labyrinth to the oblongata. Such lesions in the internal auditory meatus or at the base of the brain usually implicate other important structures and give corresponding symptoms. A not infrequent syndrome resulting from a gumma or a gummatous meningitis is a combination of auditory and facial paralysis, the glossopharyngeal and other nerves also sometimes being affected. Vertigo, forced movements, or other evidences of disturbance of equilibration indicate vestibular nerve disease or cerebellar lesion. If the glossopharyngeal nerve is involved, loss or perversion of taste may be present. Gliomatous, tubercular, syphilitic, or other growths, apoplectic and inflammatory affections, aneurisms, and perhaps other focal lesions, may involve the ventrolateral nucleus and be largely limited to it. Such affections give rise in the first place to more or less impairment of hearing, according to the extent of destruction of the nucleus. If the lesion is irritative, some of the forms of hyperacusia and paracusia may be present. When the restis or prepeduncle is involved, symptoms referable to this structure will be added to those of auditory impairment. The great spinal root of the fifth and the roots of the vestibular nerve and Deiters's nucleus may be invaded, and hence sensory phenomena in the distribution of the fifth and disturbances of equilibration may accompany the defects of audition. Lesions of the acoustic tubercle or of the trapezoid body, of the superior olive, and of the acoustic fillet give auditory symptoms. If a primary or a secondary degeneration of any of these tracts and centres is present, the symptoms are purely auditory, but if the lesions are macroscopic other important structures will probably be involved, a fact which furnishes the key to accurate focal diagnosis. Deafness,

usually bilateral, but sometimes unilateral, has been met with in a number of reported cases in connection with evidences of trigeminal disease, especially in lesions of the sensory root of the fifth. In these cases neuralgic pain has been commonly a marked and early symptom, with anesthesia later, but anesthesia is sometimes present without pain. Subjective noises have usually been present, at least until total anesthesia and deafness have developed. Deafness sometimes comes suddenly, in other cases slowly. The explanation of a case of this kind is to be found in a lesion somewhere in the pons invading the bulbocerebral tracts for hearing. As indicated on page 371, lesions of the postgeminum give auditory symptoms associated with emotional manifestations and disorders of motor incoordination, and, as indicated on page 358, the postgeniculum is probably connected with the cochlear auditory tract, and its lesions should therefore also give auditory symptoms. The affection of hearing in such a case would in all probability be associated with symptoms of thalamic disease, unless the lesion were degenerative and strictly confined to the postgeniculum. A few cases have been reported in which deafness has resulted from lesion of the internal capsule, the deafness being usually associated with hemianesthesia and sometimes with hemiplegia. In a case recorded by Dr. Spiller and the writer, deafness was present with lesion of the external capsule, but the white matter of the first and second temporal convolutions was also involved. It has been shown in several places (pages 344, 345, and 632) that lesions of the first and second temporal convolutions of the left side in their posterior portions cause word deafness, as does also destruction of the immediately subjacent white matter. A lesion of these convolutions on either side causes contralateral sound deafness.

Atrophy of the Cochlear Nerve.—The majority of cases of tabes have defective hearing, and in not a few of these the impairment goes on to total deafness. These affections of hearing may occur at any period of the disease, but they are much more common in its middle and later stages. Habermann found in a tabetic patient who had been deaf for thirteen years great atrophy of the ganglion cells in the vestibule, cochlea, and ampullæ, and atrophy of the nerve fibres as far as their termini in the brain stem; but the terminal nuclei in the bulb appeared not to have degenerated. Atrophy of the auditory nerve has been recorded in other cases of tabes. In tabetic atrophy the loss of hearing is gradual, often first in one ear and then in the other, although both may be affected at the same time. Moos and Steinbrügge, in a case of atrophy of the cochlear nerve in the first coil of the cochlea, found deafness for speech and also marked diminution of the perception of high tones. Tinnitus is sometimes present. In auditory atrophy close examination with improved means of investigation might show gradual contraction

of the general field for sound and changes in tone and pitch in special auditory fields. It should not be concluded that deafness in a case of tabes is due to tabetic atrophy until a systematic and thorough aural examination has been made. Many of the cases of tabes with impaired hearing which I have studied at the Philadelphia Hospital show lesions of the external auditory meatus, membrana tympani, and middle ear. Secondary atrophy of the cochlear nerve is of comparatively frequent occurrence, and may be the result of a variety of lesions, as of caries with necrosis, abscess, syphilitic exudates, hemorrhage, aneurisms, anchylosis of the stapes, and ossification and injuries of the cochlea. The main symptoms of secondary, like those of primary, atrophy are anacusia and tinnitus. The tinnitus is of more frequent occurrence than in tabetic atrophy, and may assume a greater variety of forms. Disorders of equilibration are also more frequent, simply because the affections leading to the secondary atrophy are usually such as to implicate more or less all portions of the labyrinth. Hallucinations of hearing have been noted in rare cases. When the cochlear nerve is secondarily atrophied as the result of compression or invasion by an extrinsic lesion, not only may the symptoms be deafness and tinnitus, but other clinical phenomena may be present, due to the action of the lesion upon adjacent structures. Such accompanying symptoms may, for instance, be convulsions, paralysis of the facial nerve, vertigo, headache, and papillitis.

Acoustic Neurasthenia.—Cases of impaired hearing with or without tinnitus, due apparently to acute or chronic auditory fatigue, are met with, and may be classed as acoustic neurasthenia. In some of these cases of acoustic neurasthenia a vertigo of mild type occurs. Pole believed that the effects of grave muscular fatigue should be particularly manifested upon the delicate special senses. He tried to determine whether physical exertion would cause any great modification of hearing, by examining the first twenty-four arrivals in a bicycle race of fifty kilometres, immediately upon their descending from their wheels, and again after a period of rest of from two to seven hours. Two acknowledged subjective tinnitus, which disappeared after repose, and nearly all showed diminution of aerial perception for the watch, while Rinne's test was negative. In a very few cases he noted some diminution in the power of perception of the high tones. With rest hearing improved, most in those who were best trained and hence the least fatigued. Another form of acoustic neurasthenia may result from the prolonged exposure of the organ of hearing to loud, disagreeable, or monotonous sounds or noises, such as are necessarily experienced in certain occupations. In such cases the auditory nerve is tired out and perhaps exhausted, just as the retina and the optic nerve may be exhausted by improper exposure to strong light and colors.

Hysterical Affections of Hearing.—The affections of hearing that accompany grave hysteria may take the form of either anacusia, hyperacusia, or paracusia. Besides deafness and increased sensitiveness to sounds, the patients may sometimes complain of acoustic impressions, which they describe by such words as gurgling, rippling, and buzzing. True hysterical deafness is usually associated with the other stigmata of grave hysteria, and especially with hemianesthesia. When hemianesthesia is complete in type, every form of sensibility may be affected ; the patient may suffer from impairment or loss of hearing, sight, smell, taste, and equilibration. It is particularly important to examine hysterical patients carefully for organic lesions of the aural apparatus.

Reflex Auditory Neuroses.—Various nervous affections with pain or discomfort as the chief feature are occasionally referable to disease of some portion of the auditory apparatus. Headache, either of slight or of severe type, may be referred to middle ear disease. A patient suffering from an affection of sight, whose acuteness of hearing is also somewhat diminished, may have the former improved by the measures taken to benefit the auditory apparatus, and the converse of this is sometimes observed. Such results are not always to be regarded as reflex phenomena, but may be looked upon as evidences of general nervous impairment or of coexisting lesions.

Etiology.—Congenital deafness is not infrequently observed among the lower grades of idiots. Occasionally there is a remarkable absence of the entire aural apparatus, as in a case observed by me at the Pennsylvania Training School for Feeble-Minded Children. In so-called deaf mutism, a term which has been objected to for the majority of cases so designated, as many of the sufferers are not necessarily mute, the pathological cause of the deficiency may be peripherally situated, or the apparent deafness and loss of speech may be dependent upon general cerebral arrest, the deficiency in the latter case being usually associated with idiocy or imbecility. The deficiency may be due to bone disease or exanthematous fevers accompanied by affections of the external, middle, or internal ear ; or it may be dependent upon limited arrest or focal lesion of those portions of the encephalon which are related to the function of either hearing or speech. Congenital or early acquired deafness is occasionally present in families of ordinary intelligence, and the tendency to become deaf is undoubtedly hereditary in some families. A peculiar form of slight or mild impairment of hearing has been observed in several members of the same family. Exfoliation of the cochlea and vestibule, and various disorders of the labyrinth, necessarily involve the neural portion of the auditory apparatus. Labyrinthine inflammation is one of the sequelæ of numerous infectious diseases. Syphilitic disease of the cochlear nerve presents itself in three forms,—as an inherited, as an acute acquired, and

as a chronic acquired affection. When deafness develops with great rapidity during infancy or childhood and without apparent exciting cause, hereditary syphilis should be suspected. Formerly many such cases were attributed in a vague way to scrofula. According to high authorities, hearing is affected in from one tenth to one third of all cases of inherited syphilis; but in these cases the cochlear nerve is not always primarily attacked. Auditory symptoms may appear with the primary, secondary, or tertiary manifestations of acquired syphilis. Anemia both of the labyrinth and of the encephalic centres for audition is theoretically possible and probable. As is well known, in cases of syncope from hemorrhage hearing is gradually lost, sounds becoming dimmer and dimmer; and associated with this gradual loss of hearing may be tinnitus and dizziness. In chronic anemia the hearing is somewhat blunted. Embolism of the vessels supplying the labyrinth may cause anemia and necrosis of the parts supplied by the vessels. The effects of quinine and the salicylates in the production of tinnitus and deafness are well known. Excessive excitement of the auditory nerve may lead to deafness, hyperesthesia, or various perversions of hearing (anacusia, hyperacusia, and paracusia). To this category are to be referred those affections which are noted among persons engaged in noisy occupations. Cases of deafness or partial deafness as the result of blows upon the head, of falls, explosions, or other accidents, not infrequently come under the care of the neurologist as well as of the otologist. In some instances the partial or complete deafness which occurs is associated with other tangible evidence of injury; but in others a total bilateral deafness may be present without such external appearances, and these are the cases most difficult of diagnosis. When unilateral or bilateral deafness is present as an isolated symptom as the result of concussion of the labyrinth, it is probable that the lesion is a hemorrhage into the cochlear portion of the labyrinth. Some light has been thrown upon the etiology of paracusis Willisii, in which the patient hears better in the presence of a noise, by experiments that have shown that increase of hearing may be produced by rapidly vibrating the cranial bones. Such vibrations doubtless increase the receptivity of the auditory nerve. Paracusis Willisii occurs with middle ear disease, the vibration of the bones of the head in these cases assisting in the conduction of sounds. Some cases of sound photisms are probably due to increased cerebral receptivity or irritability, or to unusual permeability of the tracts which associate the auditory and visual centres.

Pathological Anatomy.—Little need be said about the morbid anatomy of disease of the cochlear nerve and of the auditory apparatus in general. As the result of traumatisms, and in rare cases spontaneously, hemorrhages occur in the structure of the internal ear, and these are sometimes partially absorbed, leaving more or

less destruction of the delicate tissues of the labyrinth. Plastic exudates are probably present in most cases of syphilitic disease affecting the cochlea or any portion of the labyrinth. In chronic cases autopsies have revealed thickening of the periosteum of the vestibule, immobility of the stapes, round cell infiltration between the membranous and the osseous labyrinth, among Corti's rods and cells, and in the ampullæ and semicircular canals. Degeneration of the ganglion cells and axis cylinders of the spiral ganglion has been recorded by several observers. The deafness which results from epidemic cerebrospinal meningitis is caused, in some instances at least, by the passage of purulent matter from the meninges along the sheath of the auditory nerve. It is not necessary to speak of the morbid anatomy of tumors, aneurisms, and other focal lesions which may affect any part of the auditory pathway.

Diagnosis.—*General Remarks.* Deafness, hyperacusia, and paracusia have special diagnostic features. In studying deafness of nervous origin the existence or nonexistence of lesions in the middle and external portions of the auditory apparatus should always be first determined. In not a few cases of deafness in which disease of the central nervous system is present, examination will show that the auditory symptoms are, in part at least, dependent upon coexisting affections of either the external or the middle ear. Chronic degenerative disease may predispose to extraneural auditory affections, as when anesthesia or paresis allows changes to take place as the result of exposure. The determination of the existence of an affection of the apparatus of hearing and of the nature and exact location of the lesion causing it may require the most patient investigation. The quality and degree of the disturbance of hearing must first be decided; and in the second place a careful study of symptoms and physical signs which point to a lesion of the auditory apparatus must be made.

Remarks on the Methods of Testing for Hearing. The usual methods of investigating for hearing have been given on pages 163 and 164, but a few points not there fully discussed will be referred to here. When the deafness is due to disease of the external or the middle ear, and not to involvement of the labyrinth and nerve, hearing through the bone remains. The person tested should not see the mouth of the investigator. Each ear should be tested in turn, the one not the subject of investigation being firmly closed by the moistened finger tip or in some other way. When testing for unilateral deafness, great care must be taken to close hermetically the other ear, and as a control experiment the hearing should be tested with both ears closed. The combined hearing power for both ears, which is different from that for each ear alone, should also be tested. Speech is sometimes recognized through the mouth, the nose, and the Eustachian tube, and, to determine whether or not the patient

hears through these channels, at times during the examination the mouth and nose should be completely closed. For full details of the special methods of testing for hearing, works on otology should be consulted. The tests of Rinne, Weber, Gellé, Binge, and Politzer all have their value, and may sometimes be absolutely necessary in neurological practice. In middle ear disease, according to Politzer, high tones are generally better heard than low tones, and Lucae found that the hearing power for low toned tuning forks was much diminished, while in labyrinthine affections these low tones were often well heard. The vibrations of tuning forks of high pitch are not infrequently perceived even when considerable labyrinthine disease is present. It is important to test not only for the hearing power of single sounds, watch or clock ticks, the pitches of different tuning forks, the different ranges of high pitch produced by Galton's whistle, the direction of sounds, and the differences in tone, but also for the hearing power of the individual for speech,—his ability to hear and understand words as words. Differences in the ability of a patient to appreciate speech as speech sometimes indicate lesions or disturbances of the auditory apparatus. Not only every otologist and every neurologist but every observant person must have noticed the great difference in individuals with impaired hearing in their relative powers of appreciating simple sounds and speech. For the quantitative testing for speech ordinary conversation and whispering are both employed.

Diagnosis of Special Forms of Auditory Disturbance. The diagnosis of peripheral deaf mutism from idiocy is to be made by a thorough study of the mental condition of the child and of the evidences of physical or somatic degeneration. A careful study should be made not only of the peripheral apparatus for hearing, but also of the mental capacity and educational capabilities of the child as illustrated by its powers of attention, application, and communication through the other senses. Subjective tinnitus must be distinguished from those entotic and periotic noises which are discernible by either physician or patient, or by both, and which may not be of nervous origin. The diagnosis is to be made by a close study of the causation of such noises. In all cases the patient should be carefully studied for word deafness (see page 660). In syphilitic affections both ears are usually affected, either simultaneously or in rapid succession. Bone conduction is of course impaired. In hysterical deafness the loss of hearing is greater for conveyance of sound through the bone than for aerial conduction.

Simulated Deafness. The simulation of deafness is a subject which belongs both to clinical medicine and to medical jurisprudence, but is properly treated more at length in connection with the latter subject. Every practising physician should, however, know something of the methods of detecting unilateral and bilateral simulated deaf-

ness. Deafness may be malingered by hysterical patients for the sake of notoriety, by soldiers or sailors to evade service, by criminals to obtain indulgence or to be removed to the infirmaries or hospitals in penal institutions, and by litigants to deceive in order to receive large sums in suits for damages. I can only reëcho the statement of Gruber that the greatest circumspection is necessary in the expression of a judicial opinion with regard to the condition of the ear. In all cases of suspected simulation of deafness, even when the alleged deafness is unilateral, both ears should be carefully studied. Special works on otology and medical jurisprudence should be consulted, but a few hints and suggestions may here be given. If the normal ear is closed and a vibrating tuning fork is placed on the vertex, a simulator will be likely to assert that he does not hear, although the sound should be heard even better than when the ear is not closed. Cotton loosely packed in the ear affects very little the hearing power for speech. Various devices can be tried to deceive the supposed simulator. The detection of simulated bilateral total deafness is sometimes difficult, and is a problem which now and then confronts the general practitioner and the neurologist, as well as the otologist, particularly if the simulator is intelligent and shrewd. An anesthetic, such as chloroform or ether, can be used. When the alleged simulator is going into or coming out of the state of anesthesia he may respond to sounds, or answer questions, or in some way indicate that he hears. If a simulator can be aroused from sleep by calling, or by some form of sound or noise, the imposition may be detected; but care must be taken that the other special senses are in no way influenced, as by the vibration of the floor or bed or by the sudden admission of light into the room. Many methods of trapping or deceiving those who simulate total deafness have been suggested, but none of these will prove successful with the wary and calculating malingerer.

Prognosis.—Hysterical and neurasthenic affections have a comparatively favorable prognosis. Disease of the peripheral end organs in the cochlea is usually unfavorable, as, owing to the delicacy of these structures and the manner in which they are enclosed, they are likely to be injured beyond any possibility of regeneration if they are the subjects of inflammatory or other disease. If treated early and energetically, deafness and other auditory symptoms due to syphilitic disease of the cochlea may respond favorably to treatment. Tinnitus, unless of psychical or vascular origin, is usually a very persistent affection. The prognosis of focal lesions of the auditory apparatus depends upon the nature of these lesions and their amenability to treatment; but it must be remembered that even when a tumor, exudate, or other lesion which has caused auditory symptoms can be removed wholly or in part, it may not be possible to remedy its destructive effects upon the organs of hearing. A tumor which

has caused either blindness or deafness may be removed from the cranial cavity after optic or auditory atrophy has resulted, and deafness or blindness persist although the more active symptoms of the neoplasm have disappeared. The prognosis of deafness and tinnitus due to labyrinthine disease is relatively more unfavorable than when it is dependent upon either middle or external ear disease; it is often somewhat more unfavorable than when due to intracranial lesions, which can sometimes be benefited by active absorbent treatment.

Treatment.—When the distal terminations of the cochlear nerve and the peripheral end organs of the auditory apparatus are implicated, the treatment differs according as the affection is acute or chronic. Most acute cases are of hyperemic or inflammatory origin. Febrifuges may be required, and often leeches in the mastoid regions and free catharsis may do something towards saving the auditory end organs from total destruction. Pilocarpine used externally or hypodermatically may be of benefit in syphilitic or other cases in which rapid exudation is threatened or has taken place. Politzer suggests to replace pilocarpine by atropine if nausea and giddiness result from the use of the former. When exudation has taken place, most can be hoped for from the active use of mercury by inunction or hypodermatically, or by the administration of potassium or sodium iodide in increasing doses. When labyrinthine otitis has become subacute or chronic there is little hope of saving the auditory nerve by treatment, but the iodides may still be employed, strychnine should be given to stimulate the relics of the auditory apparatus, blisters can be applied back of the ears, and the effects of the galvanic current may be tried. For those cases of partial or complete deafness which accompany tabes and other degenerative diseases little can be done. Some transient improvement in hearing, as in other symptoms, is sometimes obtained by the use of remedies like silver nitrate, silver oxide, strychnine sulphate, and strychnine nitrate. Electricity has been recommended, but is of no permanent benefit. The underlying process is a degenerative one, and cannot be reached by this agent in the case of the auditory nerve and its ganglion any more than it can in the case of the dorsal ganglia and spinal nerves. Hysterical deafness is to be treated on the same principles as apply to hysteria in general. Here electrical application, hypnotic suggestion, the use of magnets, metallic tonics, and remedies directed to the general improvement of nervous tone are of most benefit. Among the special symptoms of disease of the auditory apparatus which call most loudly for treatment is that form of paracusia known as tinnitus. The presence of tinnitus renders life so uncomfortable, particularly to patients of nervous temperament, that the sufferers constantly appeal for a relief which, unfortunately, too often cannot be afforded. Among the modes of treatment most fre-

quently resorted to are those directed to the improvement of digestion and of constitutional states, but these are efficient in only a limited number of cases, as when some benefit is obtained from the employment of antirheumatic remedies. What has already been said about the use of remedies for the treatment of hyperemia, anemia, inflammation, and exudation is applicable also in the discussion of the therapeutics of tinnitus. Tinnitus is undoubtedly aggravated in some cases by anemia, neurasthenia, and general nervousness, and it is for this reason that the closest attention should be paid to building up the general health of the patient. Special remedies have been used empirically for the relief of tinnitus, and among these the bromides and hydrobromic acid hold a prominent place. The bromides should be given in efficient doses, usually not less than fifteen or twenty grains, and hydrobromic acid in doses of from one half to two drachms. As the hydrobromic acid needs to be largely diluted and may not agree with the stomach, the use of the bromides is to be preferred. These sedatives are most useful in those cases in which evidences of hyperemia or hyperexcitability are present. Belladonna, cannabis indica, morphine, digitalis, strophanthus, and other drugs are used in combination with the bromides for special indications. Nitroglycerin is of benefit in cases of arterial sclerosis. Coniine hydrobromate for tinnitus was given a thorough trial by Gomez, but without decided beneficial results except in one case. The largest dose given was one thirtieth of a grain, anything higher than this causing gastric disturbances, which can be avoided by taking the dose immediately after meals. Quinine and sodium salicylate, which, as is well known, produce tinnitus, occasionally prove of benefit in its treatment. Charcot suggested the use of quinine to produce cinchonism for the relief not only of tinnitus but also of the other symptoms of Ménière's disease. After the acute effects of the drug have passed away the tinnitus is sometimes found to be much lessened. Sodium salicylate can be used in the same way in doses of from ten to twenty grains three or four times daily. When anemic subjects suffer from tinnitus, preparations of arsenic and iron should be used in full doses. In the treatment of labyrinthine concussion, rest, local and general, psychical and physical, is of the utmost importance. In the early stages, local derivatives, such as leeches and blisters to the mastoid region and to the nape of the neck, are of value. When a reflex auditory neurosis is suspected, the special disease of the auditory apparatus apparently causing it should be sought for and treated. In the treatment of word deafness and other forms of cerebral deafness, after the use of remedies or surgical measures for the removal of focal lesions, such as tumor or abscess, most benefit is to be hoped for from systematic training, a subject which has been discussed when speaking of the treatment of the different forms of aphasia.

AFFECTIONS OF EQUILIBRATION DUE TO DISEASE OF THE VESTIBULAR NERVE AND ITS RELATED ENCEPHALIC STRUCTURES, AND CONJOINT AFFECTIONS OF HEARING AND EQUILIBRATION DUE TO COCHLEOVESTIBULAR DISEASE.

Functions of the Semicircular Canals.—Two at least of the great branches of the vestibular nerve originate in the ampullæ of the semicircular canals, the third in the utricle.* The ramus medius of Schwalbe, usually regarded as a branch of the cochlear nerve, goes to the ampulla of the posterior (frontal, posterior vertical) semicircular canal and to the saccule. As stated in the last section, this sacculo-ampullary nerve may be an entirely separate nerve, or a branch of the vestibular nerve. Its proper position has not yet been established. The functions of the semicircular canals, even, have not been fully determined. At one time they were regarded as of essential importance to the function of hearing; at another, as playing a minor part in this function; while the view now held by many is that they have no direct connection with audition, but are essentially related to equilibration. An analysis of all the evidence makes it most probable that these canals and the nerves originating in their ampullæ have for their special function the regulation of spatial or equilibratory impressions. Equilibration and audition are, however, probably more closely associated than any of the other senses, and the middle branch of Schwalbe may be one of the important neural factors in bringing about this association. The well-known experiments of Flourens, which have been repeated times without number on various animals, seem to prove the part played by the semicircular canals in equilibration. Briefly stated, these experiments show that when the inferior or horizontal canals are cut, movement is produced towards the right or the left according to the side on which the operation is performed; when the anterior or superior vertical canal is sectioned, the animal makes rapid movements of the head backward and forward, tending to turn a somersault from behind forward; and when the posterior frontal canal is operated upon, the animal tends to move in the reverse direction,—that is, from before backward. Section of two or of all of the canals causes complicated and bizarre movements. The results of many

* The semicircular canals, like many other organs and structures which constitute portions of the nervous system, have been variously named. The best designations are those suggested by Retzius, namely, *anterior*, *posterior*, and *external*. The anterior is also known as the superior, the superior vertical, and the sagittal; the posterior as the frontal and the posterior vertical; the external as the inferior or horizontal. While the terms suggested by Retzius are most correct anatomically, the expressions vertical and horizontal are useful particularly in describing the results of physiological experiment and of lesions of these canals.

experiments since the time of Flourens, and on the whole the results of clinicopathological observation, have tended to confirm the theory that the semicircular canals in some way act as balancing organs for the body. Some authorities, however, do not accord with this view, and a few experiments seem to contradict it. Steiner experimented on sharks caught in the Bay of Naples. The semicircular canals in these animals are readily exposed, and their excision, according to this experimenter, did not induce any disturbance of equilibrium when the shark was again placed in the water, although traction on the auditory nerves caused compulsory circular and rotatory movements. Similar experiments on frogs and lizards gave similar results. It is held by those who do not believe that the semicircular canals are organs of equilibration that the disturbances of equilibrium produced by the above mentioned experiments and lesions are due to injury of neighboring parts, as the cerebellum or its peduncles. This explanation, however, will not hold for the majority of the observations. The weight of evidence is in favor of the view commonly held, that the semicircular canals and their contents are structures concerned with the spatial sense or sense of equilibrium, although they have some subsidiary or associative auditory functions. As to the mechanism of the movements caused by operations or by lesions of the semicircular canals, some believe that they are due to irritation of the peripheral nerve apparatus; others, that they are dependent upon a withdrawal of the normal stimulus communicated by the mobile contents of the semicircular canals to the nervous structures and thence transmitted to the cerebellum and the cerebrum. Both explanations may be applicable in different cases. The membranous canals are furnished with nerves only at their ampullæ. The canals are filled with fluid. When the head is at rest the fluid in the labyrinth is also at rest, and any sudden movement of the head or body, by causing variations of pressure in the ampullæ, will call forth, through the impressions made upon the ampullary sense organs, adaptive movements of coordination. The experiments of Crum Brown have been mentioned (see page 158, Chapter II.).

Peripheral Vestibular End Organs.—The peripheral end organs of the vestibular nerve, like those of the cochlear nerve, are modified epithelial structures, to and among which pass the exceedingly fine terminal processes which are derived from the cells of the ganglion of Scarpa. The hair cells of these end organs, like those of the organ of Corti, are modified epithelial tissues. The term *crista acustica* (acoustic crest) is applied to the slight elevations which are caused by the presence of the nerve terminals and end organs in the floor of the ampullæ, and the term *macula acustica* to the terminations of the vestibular nerve in the utricle and of Schwalbe's branch in the saccule. The macula acustica differs from the crista acustica, however, in that the free surface of the neuroepithelium of the former

is covered with what is known as the otolith membrane. The otoliths (minute crystals of calcium carbonate) play some part in the reception and adaptation of the impressions received by the utricle and saccule. The ganglion of Scarpa (*intumescentia ganglioformis Scarpæ*) is situated at about the point of junction of the facial with the vestibular nerve, in or near the external auditory meatus. The cells composing this ganglion are situated along the course of both the true vestibular nerve and the sacculo-ampullary nerve of Schwalbe. Like those of the spiral ganglion, they are bipolar, the peripheral processes going to the end organs, while the central ones pass in the trunks of the nerves to the oblongata. The subdivision of the branch of Schwalbe which goes to the ampulla of the posterior semicircular canal has along its course two small ganglia of its own (*ganglia of Corti*). Other small ganglia are present on the vestibular branches to the other semicircular canals. The course of the vestibular nerve from the ganglion of Scarpa to the postoblongata has been sufficiently described when speaking of the course of the cochlear nerve (see page 710). The courses of both nerves from their termini to their end nuclei are indicated in the diagram Fig. 349.

Terminal Nuclei of the Vestibular Nerve and their Connections.—Lying ventrally to the cochlear nerve, the vestibular nerve continues as the internal root of the eighth nerve, between the restis and the descending or spinal root of the trigeminus, and terminates in three separate cell nests, usually spoken of as the mesal or chief nucleus, the nucleus of Deiters, and the nucleus of Bechterew. In contrast to the ventrolateral nucleus (or combined cochlear nuclei) the combined vestibular nuclei might be designated the dorsomesal nucleus.* Although nothing definite is known as to the distinct functions of the different nuclei which compose the entire terminal nuclei of the vestibular and of the cochlear nerve, it is not improbable that the different branches of both nerves have separate nuclear terminations in the bulb. It is of interest in this connection to note the fact that the dorsomesal or vestibular nucleus has three constituent nuclei, and that the vestibular nerve has at least three separate branches which go to different portions of the labyrinth. Eventually the separate end nuclei for each separate subdivision of

* Two of the constituent parts of this dorsomesal nucleus, like the subdivisions of the ventrolateral nucleus, have a variety of names. The dorsal or chief nucleus (called "chief" because formerly supposed to be the chief auditory nucleus) is also spoken of as *central nucleus*, *inner nucleus*, *posterior nucleus*, *nucleus of the posterior root*, and *mesial* or *mesal portion of the superior nucleus*. The nucleus of Deiters is also known as the *external acoustic nucleus*, *superior nucleus* or *lateral part of the superior nucleus*, *mesial* (or *mesal*) *nucleus of the anterior root*, *large celled nucleus*, and *inner segment of the restiform body*. The dorsal nucleus and the large celled nucleus taken together are sometimes spoken of as the *superior nucleus*. Bechterew's nucleus, an extension of the nucleus of Deiters, is fortunate thus far in not having been christened with more than one name.

the nerve will probably be determined. The bifurcation of the vestibular nerve into a descending and an ascending root after its entrance into the oblongata has been clearly demonstrated. The latter passes to the nucleus of Bechterew, and from this enters the acoustic cerebellar bundle, giving collaterals which pass not only to the dorsal or chief nucleus but to the nucleus of Deiters, also distributing other finer fibres which pass to the cells lying between the fibres of the bundle of the descending root. The process of the peripheral "neuron" or nerve cell arises in the ganglion of Scarpa and splits up about the cells of the dorsal nucleus and other nuclei as just described. One of the bifurcating portions passes into the descending and the other into the ascending cerebellar root. The next neuron, beginning in the dorsal nucleus, passes in the trapezoid body, crosses in the raphe to the mesal fillet, and thus continues towards the cortex. Bruce found that in transverse dorsoventral sections made at the extreme cephalic limit of the postoblongata through this structure and the flocculus, some of the fibres of the flocculus appear to bend upward towards the cells at the lateral angle of the oblongata, in a position which corresponds to that given by Bechterew as that of one of the nuclei of the vestibular roots of the auditory nerve (Bechterew's nucleus). The fibres at this situation, according to Bruce, break up into a fine network, from which a comparatively small number passes backward along the lateral wall of the fourth ventricle towards the vermiform lobe. Their manner of termination he was not able to establish. The flocculus and some portions of the vermis would therefore seem to be important central (cerebellar) connections of the nuclei of the vestibular root of the eighth nerve. The same investigator, as already stated, has shown it to be also a central (cerebellar) connection of the accessory nucleus of the cochlear nerve, and almost certainly of the sixth nucleus. As summarized by Ramón y Cajal, by means of the ascending branch the vestibular nerve is spread out over the upper portion of the nucleus of Deiters, over the entire nucleus of Bechterew, over the multipolar cells of the acoustic cerebellar bundle, and finally over the tegmental nucleus and dentatum of the cerebellum. The cells of the chief or dorsal nucleus are connected with the lateral vestibular tract, in which they unite with fibres coming from the nucleus of Deiters. A large portion of the axis cylinders of the nucleus, however, cross the raphe and enter the lateral vestibular tract of the opposite side. As stated on page 82, the acoustic connections of Deiters's nucleus have been regarded by many as doubtful. The most recent researches, and especially those of Ramón y Cajal (1896), show the important relations of this nucleus to the vestibular nerve.

Cerebral Terminus of the Vestibular Nerve.—It is probable that the cerebral representation of the vestibular nerve is distinct from that of the cochlear, and Dana has suggested that this

representation is in the temporal lobe, and especially on the right side, basing this view upon two personal cases of focal lesion of the right temporal lobe, and also upon a study of the meagre literature of the subject. Bechterew believes that the evidence afforded by the cases compiled by him justifies the hypothesis that the prepeduncles are connected with the upper portion of the parietal lobe, but Dana holds that, while the muscular sense may be impaired through destruction of this lobe, the true spatial sense is not affected. Schäfer has shown that irritation of the temporal lobe causes conjugate deviation of the head and eyes. The well known fact that a large proportion of deaf mutes cannot be made vertiginous has some bearing upon this subject. Bechterew and others believe that they have shown that the temporo-occipital region is connected with the cerebellum of the opposite side by a band of afferent fibres which pass by way of the medipeduncle, the pontile nuclei, and an external bundle in the crus. That this is not connected with the first and the upper and anterior part of the second temporal convolution was shown by Spiller and the author. In a case of cerebral abscess situated at the posterior part of the external capsule, and involving the medullary substance of the first temporal and part of the second temporal convolution and also the posterior part of the lenticula, this band of fibres was not degenerated. Dejerine has shown that the external bundle of the crus is formed by projection fibres which come from the temporal lobe, and especially from the second and third temporal convolutions, and he also believes that this fasciculus remains intact after lesion of the occipital lobe, differing in this respect from Bechterew. In the "naming centre" case (page 345), in which a tumor involved the midtemporal region and chiefly the left third and fourth convolutions and subjacent medullary substance, one of the first symptoms complained of by the patient, five years before her death, was vertigo; and this was a recurring phenomenon. She did not, however, suffer from ataxia or forced movements. In Dana's two cases destructive lesions were present in the middle portion of the right temporal lobe, and in both vertigo and forced movements were important symptoms. It must not be overlooked that in one of these cases, at least, the dura was markedly involved, and that vertigo is a well known symptom of irritative dural disease. The membranes were not, however, involved to any extent in the other cases, and special forced movements are not characteristic of pachymeningeal vertigo.

Clinical History.—*Symptoms of Pure Vestibular Disease.* The special symptoms of disease of the vestibular nerve and of its central connections are, speaking in general terms, disorders of equilibration: these symptoms are usually more or less severe vertigo and a sensation of unbalanced movements, which the patient may refer to himself. Often the chief complaint is of dizziness, giddi-

ness, or vertigo; sometimes, but less frequently, it is of pitching, reeling, swaying, or staggering. One or several of these terms may be chosen by the patient to describe what to him seems to be his most important symptom. The terms vertigo, giddiness, and dizziness are commonly used interchangeably, or without any special distinction, by doctors and patients. Vertigo comes from *vertere*, "to turn," and the word is best used to define a sense of disturbed equilibrium which may or may not be associated with actual movement of the patient or of objects external to him. It is not merely a sensation, but a sensation which indicates that a motor process has been disturbed. Occasionally cases of pure vestibular nerve disease are observed. Such cases have fallen under my own observation. In them vertigo, with a marked tendency to fall or turn in a certain direction, has been present, without any disturbance of hearing, and the cases could not be explained on the theory of disease of the cerebellum or of any portion of the brain. The following cases belong to this category. A woman, thirty-four years of age, had at intervals since her fourteenth year been subject about the menstrual period to slight attacks of vertigo. Several weeks before coming under my observation this "vertigo" had returned, and had persisted. Headache was absent, and careful examination revealed no loss of hearing and no aural disease. She showed an almost constant tendency to go to the left, complained of her head swimming and of objects seeming to move. The feeling recurred whenever she turned to the left. It was necessary for her to take great care in getting off street cars, for if she got off towards the left she was liable to fall. She was tested a number of times, and always tended to fall to the left on turning in that direction, while when she turned to the right there was no tendency to fall to that side. She had no condition of refraction sufficient to account for the trouble. A middle-aged physician consulted me with reference to a persistent vertigo. Six months previous to coming under observation he had his first attack, one week later the second, and two months before my examination he had a third attack, and afterwards was almost constantly vertiginous, the intensity of his symptoms varying. He had a tendency sometimes to go to the right, at other times to the left; on looking up to the left he inclined to the right, and the reverse. Sometimes he almost fell. He had no tinnitus and no auditory symptoms of any sort. Careful examination of his eyes showed some astigmatism. He presented no paralytic symptoms. In the purer forms of vestibular vertigo the patient, without any auditory symptoms, may show a tendency to turn or fall in some particular direction, or objects may seem to be falling or moving, or a tendency to deviate to one side in standing or walking may be shown. While vertiginous paroxysms are nearly always most marked when the patient is in a standing or a sitting position, this is not always the case. Both in vestibular vertigoes

and vertigoes of the purer forms and in those due to cochleovestibular disease (Ménière's disease) the paroxysms of dizziness come on occasionally when the patient is in the recumbent position. They may occur at night after he has retired. It is not impossible in such cases, as has been suggested by Gowers, that the tendency of the attack to occur only when the patient is in the horizontal position is related to the fact that the focus of disease is in some one particular portion of the semicircular canals.

Special Symptoms of Focal Lesions in the Vestibular Pathway. A lesion limited to either the vestibule or the semicircular canals causes vertigo, and this in most instances is extreme. It varies in character and severity according to the extent and the exact location of the disease. The directions taken in the sensations of falling and in the tendency to fall differ according to the side of the body and the particular canal most markedly affected, in accordance with physiological observations already discussed. If a gross lesion is situated in the internal auditory meatus or on the lateral border of the oblongata, the symptoms may show implication not only of the cochlear nerve but also of the facial and of the pars intermedia of Wrisberg. The remarks made when focal lesions of the cochlear nerve were discussed are applicable here. Bruce has suggested that tumors of the dura situated near the internal auditory meatus probably give rise to vertigo through their influence on the flocculus and the vestibular fibres which run in its peduncle. A lesion affecting the nerve after it has passed into the oblongata produces symptoms which vary according to its exact position and its ramifications. Auditory, sensory, motor, vasomotor, and other symptoms may be present according to the degree of involvement of the cochlear nuclei, the fillet, the pyramidal tract, and other structures. Focal lesions in several situations may cause conjointly disturbances of equilibrium and of vision. The abducent, optic, oculomotor, and eighth nerves, including both the cochlear and the vestibular nerve, have important connections with one another, and with various centres and tracts, many of which have been described.

Tabetic Atrophy of the Vestibular Nerve.—According to Althaus, the vestibular portion of the eighth nerve suffers more frequently than the cochlear nerve in tabes. I have made a number of observations on the occurrence of vertigo in tabes. The patients in these cases experience a sensation of fulness and swimming in the head; they may feel as if everything is spinning around, and may stagger and lose their balance unless supported. A tendency may be shown to fall in some special direction,—backward or forward, or to the right or the left. In these cases it is not improbable that the nuclei of origin or the bulbar nuclei of the vestibular nerve are attacked by an irritative and degenerative process similar to that which occurs in the spinal ganglia.

Vertigoes not directly the Result of Disease of the Apparatus of Equilibration.—*Classification of Vertigoes of Indirect Origin.* Thus far in the present section, in our consideration of disorders of equilibration, the symptoms discussed have presumably been due to lesions or disturbances directly affecting that portion of the labyrinth from which the vestibular nerve originates, or the trunk, root fibres, terminal nuclei, bulbocerebellar or bulbocerebral tracts, or cortical termini of this nerve. The equilibratory apparatus may, however, be pathologically influenced by causes not acting primarily on any of these parts, and in this indirect way some of the best known forms of vertigo are produced. These vertigoes are considered under various heads and from different points of view by different authors, but their discussion properly belongs here, as in their production the apparatus of equilibration must take part. The most important of these varieties of vertigo are (1) reflex vertigo; (2) vertigo dependent upon irritative intracranial disease situated outside of the equilibratory apparatus; (3) vertigo due to disease of the heart and bloodvessels; (4) vertigo which has its source in the state of the blood, this probably including (5) Gerlier's vertigo; and (6) vertigoes or pseudo-vertigoes of functional or mixed origin.

Reflex Vertigo. Irritation may be reflected to the apparatus of equilibration from almost any portion of the body, near or far. It is for this reason that special designations are sometimes given to particular varieties of reflex vertigo, as when they are termed ocular, aural, nasal, pharyngeal, gastric, gastrointestinal, hepatic, uterine, or ovarian, according to the organ which seems to be the fountain head, but if they are truly reflex disorders the mechanism of their production is practically the same in all cases, namely, a disturbance of equilibrium produced by the reflection of an abnormal stimulus upon a more or less nonresisting equilibratory apparatus. The reflex origin of vertigo is sometimes more apparent than real, and too great a tendency is shown to attribute this, as well as to refer other nervous symptoms, to reflex action. Not a few of the cases regarded as reflex are probably due either to a toxic state of the blood or to some undetermined condition directly acting upon the vestibular nerve apparatus. It remains true, however, that a certain percentage of cases can be properly classed as reflex vertigoes and are to be treated by attacking the primary focus of irritation wherever it may be situated. The occurrence of vertigo, usually in the form of a transient giddiness or dizziness, as the result of disease or disturbance of the external or middle ear may or may not be regarded as of reflex character, according to the peculiar method of its development. It is only necessary to remember that a true aural vertigo may occur even when the labyrinthine portion of the neural apparatus for hearing and equilibration is intact. Every one is familiar with transient vertigo from irritation or compression of the tympanic membrane

and from Eustachian or other forms of middle ear disease. In these cases the labyrinthine structures, cochlear and vestibular, may be affected, either through the transmission of pressure, by continuity of inflammation, or by reflected irritation. The vertigo is to be relieved by measures directed to the parts primarily affected. Transient vertigoes of external origin often demand earnest attention, and in most cases are probably conjointly toxic and reflex. These are the vertiginous seizures which arise from the ingestion of articles of food or drink which are indigestible or with regard to which the individual has special idiosyncrasies. The vertigoes which are to be traced to nasal polypi, to irritation of the gums, to tapeworm, and to various other special forms of irritation need only this brief reference. One form of laryngeal vertigo is sometimes described as an "epilepsy," and is, because of its serious appearance, of especial clinical importance. It is usually a disease of middle age, although it may occur at any period of life. The sufferer from the attacks frequently has some form of chronic laryngitis or bronchitis. The paroxysms begin with unpleasant sensations, often those of burning or tingling in the larynx or trachea, with dyspnea and an uncontrollable spasmodic cough. The patient may fall and remain for a brief time unconscious, and often twitchings or light convulsive movements have been observed.

Interrelationships of Vertigo and Ocular Disturbances. Affections of ocular centres and tracts, organic or functional, may excite auditory and vestibular symptoms; and when the vestibular nerve, its centres, or its associating systems are affected, important ocular symptoms, as well as disorders of equilibration, may be present. Vertigo, on the one hand, may be accompanied by ocular manifestations like nystagmus and vacillating strabismus, and, on the other hand, refractive, accommodative, and other visual defects and disturbances may reflexly cause vertiginous manifestations; and, further, a vertigo which is primarily dependent upon a lesion of either the peripheral end organs, the trunk, or the central pathway of the vestibular nerve may be intensified by errors of refraction. Such refractive errors may remain unnoticed until the development of some disease of the apparatus for equilibration. While vertigo may be caused solely by ocular defects, such cases are rare. Weir Mitchell and Thompson were among the first to call attention to the subject of vertigo due to eyestrain. Cases have been recorded by Bonnier, Osler, and others, showing the special relationship between labyrinthine vertigo and ocular defects. The facts necessary to the understanding of this subject have been given in previous paragraphs in the discussion of the nuclei and tracts of the vestibular and cochlear nerves and their associated structures. In one of Bonnier's cases disturbance of accommodation followed auditory irritation, the latter having been caused by aural irrigation which drove a plug of wax

against the tympanic membrane. A patient of Osler's for eighteen months before coming under observation had had a number of attacks of vertigo, with flatulence, a few of them severe. The vertigo appeared to be both objective and subjective, objects seeming to go to the right, and the patient feeling as if he also turned. The man was a little deaf, particularly in the right ear, in which he had almost constant tinnitus. Examination showed that the deafness was probably due to labyrinthine disease. Decided hyperopic astigmatism was also present. Properly adjusted glasses improved the patient's vision, and relieved his vertigo for more than two months, when he began to suffer with vomiting, which continued for two weeks until his death. It seems probable that the primary lesion in this case was somewhere in the cochleovestibular apparatus. The case is of particular interest as showing the intimate and reciprocal relationship between the ocular, the equilibratory, and the digestive apparatus. Double vision may occur during or after an attack of aural vertigo, and in addition objects sometimes appear to undergo a jerking movement. The patient may be unable to calculate distances properly, and so cannot readily touch an object, or in walking may miscalculate and step beyond an intended point.

Vertigo due to Irritative Intracranial Disease, and especially to Brain Tumor. Vertigo may be dependent upon irritative intracranial disease, and especially upon tumor and pachymeningitis, not referring in this connection to the disturbances of equilibration arising from disease of the cerebellum or the quadrigeminum. The three most frequent general symptoms of intracranial tumor are headache, nausea or vomiting, and vertigo, and these are commonly dependent upon the same mechanism. Most cases of brain tumor originate in the encephalic membranes. The trigeminal nerve has a wide distribution in the dura, and intense localized irritation of its branches gives rise directly to pain, and indirectly to vertigo, nausea, or vomiting. The reflection or irradiation of powerful impressions from the bulbar nuclei of the fifth to the vestibular and pneumogastric nerves is the best explanation of the mechanism of the production of these symptoms. The fact that a vertigo is due to a neoplasm, and not directly to disease of the vestibular nerve, can be best determined by a study of the general and focal symptoms of tumor of the brain. The remarks just made regarding the vertigo of brain tumor are equally applicable to that observed in localized meningitis.

Vertigo due to Disease of the Heart and Bloodvessels. Vertigo is sometimes dependent upon imperfect action of the heart, and this itself may be due either to degenerative disease of the cardiac walls or to faulty innervation. The brain in such instances is irregularly and imperfectly supplied with blood. Vertigo may also be due to disease of the bloodvessels, as to arteriosclerosis (see pages 445 and

446). The diagnosis of these cases as there indicated is to be made chiefly by a careful examination for arterial or arteriocapillary fibrosis, and the usual accompanying conditions of the heart, kidneys. liver, and other organs.

Vertigo due to States of the Blood. Vertigo may have its source in the state of the blood, as when it arises from anemia or hyperemia, lithemia, and a number of toxemias, including those occurring in connection with infectious diseases and from the direct action of drugs and poisons. Lithemic vertigo is of great practical importance, as it is of comparatively frequent occurrence, usually excites great alarm, and is more or less rapidly relieved by treatment directed to the lithemic state. Occasionally a pronounced vertigo is one of the early symptoms of an acute infectious disease, and it may be accompanied by a severe headache, or even by marked mental symptoms. The vertigoes due to the ingestion of alcohol or of special drugs are much influenced by individual idiosyncrasy. The dizziness due to opium and some other narcotics and drugs is usually made much worse by the erect position, and often disappears entirely when the patient is lying down.

Gerlier's Disease, or Paralyzing Vertigo. Under the name of *paralyzing vertigo*, Gerlier of Ferney has described an affection, probably of toxemic origin, of which little has been observed in other parts of the world than southern France and Switzerland. The affection comes on abruptly, and its phenomena recur in paroxysms. The patient becomes very dizzy, has severe pain in the neck, double ptosis, a weakness of the muscles of a paretic or a paralytic character, general lassitude, and in some cases loss of speech. Consciousness is not lost, and the attack commonly passes off in a few minutes. The first attack is followed by others which occur at intervals of a few months, weeks, days, or hours; most frequently they are weeks or months apart, and the general health of the patient in the intervals is usually good. This curious disease has been noted especially in the canton of Geneva, where it is sometimes epidemic. It occurs more frequently during the hot weather, and chiefly among laborers, herdsmen, or others working on farms. It is believed by Gerlier and others that the affection is due to germs from the marshes and stables, but, as suggested by Hirt, this does not explain the immunity of the female sex. Thus far nothing has been learned of the pathology and morbid anatomy of this disease.

Vertigoes of Functional or Mixed Origin. Certain forms of vertigo or pseudovertigo have been described as mental or psychical, but should in the majority of cases be considered rather as obsessions. The vertigo attributed to hysteria and neurasthenia can usually be explained better by ocular defects, changes in the blood, reflex irritation, psychical influence, or labyrinthine complications. In like manner the so-called essential vertigo which has been described by

Ramskill, Weir Mitchell, and others recedes more and more as knowledge increases. The vertigoes with nausea and vomiting which so frequently arise during journeys by sea or rail are to be arranged under one or several forms already described; probably they are commonly dependent upon direct or indirect disturbances of the contents of the labyrinth. They are sometimes spoken of as *mechanical* vertigoes, and to the same class are to be referred those forms which result from sudden or unusual movements, as from swinging and turning rapidly, or from the motion of elevators.

Cochleovestibular Disease.—The effort has been made in the preceding pages to consider separately affections of the cochlear nerve and of the vestibular nerve. It has been shown that these nerves originate in separate portions of the labyrinth, that they pass separately although for a part of their course in the same bundle to the oblongata, that they separate on entering the oblongata into different roots, that they terminate in different bulbar nuclei, that their bulbar connections and bulbocerebral pathways are different, and that they probably have different cerebellar and cerebral termini. It has also been shown that a different and separable symptomatology can be recognized for each of these nerves and their related structures. While this is true, it is a well known clinical fact that the apparatus for hearing and that for equilibration are frequently affected together, perhaps almost as frequently together as separately. The clinician is confronted with many instances of cochleovestibular disease,—disease which may affect all or various parts of the labyrinth, the conjoint cochlear and vestibular nerve trunks or nerve roots, or the centres and pathways of both nerves within the brain stem and the brain. These points have been already sufficiently emphasized in the consideration of each of these nerves, with the exception that it is necessary to discuss one or two well known affections in which conjoint disease of the labyrinthine portions of both nerves is present. One of these is the labyrinthine otitis of Voltolini; but the most important is that which has long been known as Ménière's disease.

Voltolini's Labyrinthitis.—Various forms of otitis labyrinthica are known to otologists, and for their full discussion works on aural disease should be consulted. As, however, both cochlear and vestibular nerves are necessarily involved in these affections, and as the neural and encephalic symptoms sometimes dominate the clinical picture, it is important that some at least of these affections should receive consideration from the neurological standpoint. Voltolini, against much opposition, advocated the theory that a primary labyrinthitis was of comparatively frequent occurrence, and his views are now generally accepted, although, doubtless, some of the cases attributed by him to primary inflammation of the labyrinthine structures were secondary to disease elsewhere. The disease is of most fre-

quent occurrence among children, but cases in adults with autopsies have been recorded. In some of its features the disease closely simulates meningitis. Meningitis may indeed occur secondary to primary labyrinthine inflammation, and the reverse is well known to occur. Most frequently the labyrinthitis begins in children abruptly, coming on with fever and excitement, and sometimes with vomiting; unconsciousness, partial or complete, may ensue early. In a common type of the affection in from two to four days the unconsciousness disappears and the children recover their senses rapidly, but when they first attempt to walk are found to stagger. This gait gradually disappears, but incurable deafness is left. Considerable differences are observed in different cases; occasionally fever is absent entirely or almost entirely throughout the attack. The diagnosis of labyrinthitis is often difficult, and is to be made by a close study of the evidences of aural and vestibular disease. Its recognition is important, because while the prognosis as to hearing is bad it may for life and subsequent general health be comparatively good.

Ménière's Disease.—*Ménière's Syndrome and its Pathological Cause.* The manner in which Ménière summarized his subject showed the probability of the existence of nerves with distinct functions taking part in the production of the syndrome which bears his name. He endeavored to establish that a previously normal auditory apparatus may suddenly become the seat of functional disturbance, consisting of noises of a variable nature, which were accompanied sooner or later by diminution of hearing; functional troubles which might give rise to vertigo, unsteadiness of gait, turnings to right or left, and falling, these being sometimes attended by nausea, vomiting, and syncope. These manifestations, often intermittent, culminate in deafness, which gradually grows worse and becomes total. Ménière held that the lesion which was the cause of these disturbances was situated in the semicircular canals, not making any clear distinction between the nerve distribution in the cochlea and in the semicircular canals. Ménière's syndrome in its most complete and independent form is without doubt most frequently of labyrinthine origin; but, while aural vertigo usually means labyrinthine vertigo, the labyrinth is not always the seat of the primary lesion, and in some cases no lesion of the latter is present, irritation being reflected to it, or inflammation extending to it, from other parts. It is best, therefore, to consider aural vertigo under the three forms of external ear vertigo, middle ear vertigo, and internal ear vertigo, according as the irritation which produces the symptom is in either of these portions of the ear; and following this classification the term Ménière's disease, if retained at all, should be used to indicate vertigo and its accompaniments due to disease or injury of the labyrinth. It is a disease in which both hearing and equilibration are affected as the result of lesions implicating the peripheral expansion

of both the cochlear and the vestibular nerve. Gruber has suggested either to apply the term Ménière's disease solely to those cases in which a hemorrhage has occurred in the labyrinth, or else to drop it altogether; and most authorities are inclined to restrict it to an acute or rapidly developed disease of the labyrinth. In Ménière's original case the lesion was a reddish plastic exudation in the mucous membrane of the labyrinth, no other evidence of disease being present. Any sudden effusion or exudation upon or into the membranous labyrinth will interfere with and probably annihilate its functions. A disorder which may be properly classed as Ménière's disease usually presents itself in two forms: (1) a transient but severe apoplectic affection; and (2) a chronic affection of varying severity with marked paroxysms or exacerbations.

Transient Apoplectic Form of Ménière's Disease. In the abrupt apoplectiform variety of Ménière's disease the patient, in previously good health, is suddenly seized with symptoms which sometimes so closely simulate a true cerebral apoplexy as at first to deceive the expert diagnostician. Such acute apoplectiform cases are usually of bad omen so far as hearing is concerned, although in rare instances the patient may entirely recover, probably because of the rapid absorption of a hemorrhage or other effusion. Kenefick has reported such a case. The patient, a man aged forty-five, with no history of previous disease, was attacked suddenly in the night with violent vomiting and persistent and alarming dizziness. He showed no motor nor sensory disturbance. Noises in the right ear and marked deafness were present. The vomiting and dizziness continued for several hours, then ceased, but began whenever he raised his head. The vomiting yielded to ipecac in small doses, but the dizziness and deafness continued. Visual hallucinations were present, but disappeared in a few hours. The slightest attempt to rise gave the patient the sensation that the bed and himself were being rapidly revolved. Vomiting ceased, but the deafness remained two weeks, when this and the giddiness gradually disappeared. At the end of six weeks he was able, with tottering gait and with assistance, to reach his office daily, and finally he completely recovered.

Paroxysmal Form of Ménière's Disease. In the majority of cases Ménière's disease is a subacute or chronic affection, which appears first about middle life or a little later. The disease, even when it assumes the chronic form, sometimes begins with an abrupt attack. The paroxysms or exacerbations of the disease may recur at very irregular intervals; sometimes several succeed one another in quick succession, giving a form of vertiginous status. In others a considerable interval may intervene between the different attacks; in still others the patient is liable to be attacked at almost any time, especially if he is not careful to avoid haste and excitement. In the paroxysms the tinnitus and the vertiginous sensations may be

extreme, nausea or vomiting may or may not be present, and the deafness, if not already complete, is increased. Occasionally forced movements and ocular manifestations, such as diplopia, nystagmus, and visual hallucinations, are present. In rare instances unconsciousness results, but this is momentary. A sense of terror usually accompanies the attack, and the patient is left pale, and perhaps covered with profuse perspiration. In the intervals between the attacks, which may vary considerably in severity, the patient usually suffers from a train of symptoms—more or less deafness, tinnitus, and slight vertiginous sensations—which indicate implication of the cochleovestibular neural apparatus. Not infrequently the symptoms, as regards both the paroxysms and the intervals between them, progressively advance, but when deafness becomes complete the vertigo may disappear.

Etiology and Pathology of Vestibular and Cochleovestibular Disease.—In many particulars the etiology of disease of the vestibular nerve is the same as that of cochlear disease, and for numerous points of causation it is only necessary to refer to what has been said under etiology in the previous section (see page 721). Disorders of the vestibular nerve or of the peripheral cochleovestibular apparatus may, for example, be congenital, or may be due to exfoliation of the bony labyrinth, to labyrinthine inflammation of various types, to local syphilitic disease, to anemia, hyperemia, or toxemia, to extravasations into the labyrinth, and to blows, falls, and other traumatisms. Exposure to high temperature sometimes plays a special part in the production of forms of labyrinthine inflammation. One of my patients was overcome with heat while working in his office on a hot summer day, the attack coming on with faintness and dizziness; he was so weak and giddy that for two weeks he was confined to the house, and ever since, now more than a year, he has been much troubled with a sense of vertigo, which is not excessive, but seldom leaves him. He usually feels as if going to the right. Hearing is defective in both ears, but more markedly so in the right than in the left, and careful examination shows that the defect is due to labyrinthine disease. Any form of intracranial focal lesion affecting the vestibular nerve or its central terminations and continuations will give rise to disorders of equilibrium, a subject to which sufficient reference has been made when discussing focal lesions. The etiology of the transient, apoplectic form either of pure vestibular vertigo or of Ménière's disease is comparatively simple. It is commonly due either to intense hyperemia or to a hemorrhage or other effusion into some portion of the labyrinth. The subacute or chronic paroxysmal form of Ménière's disease may in different cases certainly have a different pathogenesis. It cannot be doubted that in not a few instances the paroxysmal and continuous symptoms of Ménière's disease are due to some form of chronic labyrinthine disease, most

commonly a labyrinthitis, and this may be due to syphilis, tuberculosis, or other infection, or may have originated from some special cause, as from a traumatism. In these cases the patients suffer more or less constantly from symptoms which indicate cochleovestibular disease, and the acute, irregularly occurring paroxysms are probably dependent upon temporary changes in the circulation. An interesting vasomotor theory has been advanced by Brunner to account for some of the cases of Ménière's disease. A toxic condition of the blood, an irritative focal lesion, or some other initial cause acting upon the vasomotor nerves of the labyrinth leads to paroxysmal disturbances of the circulation, and to variations of labyrinthine pressure, during which the patient suffers from the symptoms which make up the picture of Ménière's disease. The underlying cause of the disease as understood by the author is situated somewhere in the labyrinth. In making this statement he is fully aware that in not a few cases the Ménière's syndrome may in whole or in part result from extralabyrinthine encephalic disease, but these cases should not be regarded as true instances of Ménière's disease. In a case recorded by Wolfe in which Ménière's syndrome was present, autopsy showed a tumor of the amygdala and another of the cerebral cortex. Nothing more regarding the morbid anatomy of the affections under consideration need be said than has been given under various preceding heads.

Diagnosis.—The manner in which disease of the cochlear and the vestibular nerves and their correlated central structures has been discussed in the preceding pages renders it unnecessary to discuss at any length the subject of diagnosis. The differential features of cochlear, vestibular, and cochleovestibular disease have been largely given in discussing their symptoms; and the differentiation of the etiological varieties of vertigo has been sufficiently indicated. One of the most important diagnostic matters is to distinguish between an acute encephalic apoplexy and the transient but severe apoplectic form of Ménière's disease. This distinction is sometimes difficult, and a mistake is more likely to be made because of the fact that the apoplectic form of Ménière's disease is rare, while apoplexy of the ordinary type is comparatively common. Dizziness and vomiting are of somewhat frequent occurrence preceding apoplectic attacks, but in some cases tinnitus and other disorders of hearing may also be prodromes, as for instance when the affection occurs in the auditory sphere of the cerebrum. When a sudden attack of partial or complete deafness occurs in an individual whose hearing has been previously normal, associated with the phenomena of an apoplectic attack, and when later the gait becomes uncertain or staggering but without paralysis in the areas of other cranial nerves than the eighth, and when an examination made soon after the attack shows a normal tympanic membrane and permeable

Eustachian tube, the conclusion that an affection of the labyrinth is present can be reached with great probability. The reader is referred to the section on cerebral hemorrhage (pages 465–469) for further diagnostic points. The diagnosis of Voltolini's labyrinthitis can be made only by a close study of the symptomatology of the cases. It occurs most frequently in children, and it is sometimes endemic or epidemic, facts that are of diagnostic value. The diagnosis of Gerlier's vertigo is to be made from Ménière's disease and other forms of vertigo, and from syphilitic, alcoholic, and other varieties of apoplectic attacks. The symptoms and course of the disease as detailed are so absolutely distinctive that a mistake is not likely to be made, particularly if the affection occurs among farm hands and is endemic. Seguin has called attention to the close similarity of the symptoms to those produced by poisoning by conium maculatum.

Prognosis.—The prognosis of disease of the vestibular nerve and of the cochleovestibular apparatus must of course depend upon the cause or special variety of the disease. When due to hyperemia or to acute inflammation, the prognosis is relatively good. When dependent upon chronic inflammatory disease, it is usually bad, although something can be done in such cases by persistent treatment. The prognosis of the different forms of vertigo of indirect causation is dependent upon the practicability of relieving or removing the source of the irritation. In rare instances, as in Kenefick's case to which reference has been made (page 741), almost complete recovery takes place from acute apoplectic Ménière's disease. In the vast majority of cases, however, the damage which has been done can be remedied to only a slight degree, if at all. The prognosis of Voltolini's labyrinthitis, or of labyrinthitis secondary to extralabyrinthine disease, is usually bad so far as the affection of hearing is concerned. Considerable improvement is sometimes made in the disorder of equilibration,—which indeed is more likely to persist if the deafness is incomplete. The prognosis of the paroxysmal form of Ménière's disease is variable. It is bad so far as complete recovery is concerned, but marked improvement may take place, and in rare instances the paroxysms cease. On the contrary, however, the disease may become so severe and the paroxysms recur with such frequency and suddenness that the patient is compelled to take permanently to bed. The prognosis as to final recovery in Gerlier's disease is good, but the patient may have a succession of attacks. Great alarm is usually felt by the sufferers and their friends.

Treatment.—Whatever measures of treatment are decided upon for labyrinthine disease, whether it takes the form of cochlear, vestibular, or cochleovestibular manifestations (Ménière's disease), the indications for treatment are various, and differ in different cases. Nearly all that has been said regarding the treatment of cochlear disease is applicable here, and need not be repeated (see page 726).

The local conditions present must in all cases be thoroughly considered. As syphilis is a somewhat frequent cause of Ménière's syndrome, and as even when it is not a part of the history the disease may be hemorrhagic or inflammatory, remedies like the iodides and mercury are often distinctly indicated, and sometimes their administration is followed by great benefit. When the rheumatic or the gouty diathesis is present, cathartics, alkalies, the salicylic compounds, colchicum, and lithia salts, should be given a thorough trial. Charcot strongly advocated the use of quinine, as stated when discussing the treatment of cochlear nerve disease. In connection with the use of large doses of quinine it is worth while to remember that we have a well known but not very common form of vertigo which is distinctly malarial in origin, the toxic agent probably acting upon the nervous apparatus of the labyrinth or upon the encephalic centres of equilibration. Little is known as to the best methods of treatment of Gerlier's disease. In a case recorded by Ackerman the attacks disappeared during the employment of warm baths and two grammes of potassium iodide daily. Although Ménière's disease is either primarily or secondarily labyrinthine, when the indications for treatment are being considered by the physician the importance of the central condition—of the state of induced instability of the cerebellar or other centres of equilibration—should not be overlooked. Remedies like the bromides which are efficient in the reduction of cortical or ganglionic excitability should be used. Arsenic may be combined with the bromides, both to prevent bromism and for its roborant effects on the nervous system; and belladonna, aconite, antifebrin, antipyrin, and phenacetin may also be tried for their effects on the centres and the circulation. The use of strychnine or nux vomica has many supporters. A method of treatment made use of by Hamilton in one case is suggestive. The patient was in the habit of asking his wife to turn him the other way when the attacks of vertigo occurred, and as a portion of his treatment the doctor had him practise at intervals turning in a direction opposite to that caused by the disease. This procedure and other methods of balancing gymnastics may prove of service in a few cases. Local counterirritant or derivative treatment has sometimes a basis for its use, and great benefit has followed such measures as counterirritation or leeching behind or below the ears, and the application of the cautery over the mastoid. The treatment of the miscellaneous forms of vertigo of indirect origin—those due to reflex causes, ocular disturbances, intracranial affections, diseases of the heart and bloodvessels, states of the blood, hysteria, and neurasthenia—are to be treated by measures directed to the relief of the causes, the most important of which measures have been referred to when considering the vertigoes themselves in preceding paragraphs of this section.

AFFECTIONS OF SIGHT DUE TO DISEASE OF THE OPTIC NERVE AND OF THE ENCEPHALIC VISUAL APPARATUS.

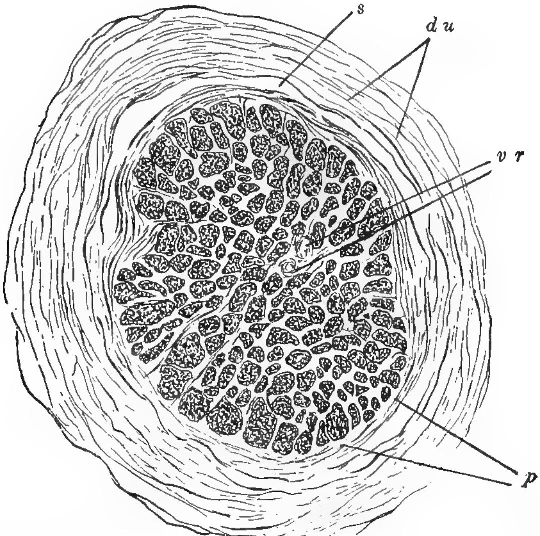
The Optic Nerve a Part of the Central Nervous System.—It has already been stated that the olfactory nerve and bulb are now generally regarded as integral parts of the forebrain. It is also held by many that the optic nerves and tracts should be recognized as a part of the central nervous system, as much as the cerebellum or the posterior columns of the spinal cord, to which they present a close similarity. Like the posterior columns, the optic nerves grow out of cells in the direction in which they convey impulses. They do not have the sheath of Schwann, as do true peripheral nerves. The optic tracts, the parts internal to the chiasm, in structure and in the arrangement of their constituents resemble the white matter of the brain. Their myelin fibres are intermingled with neurogliar cells. The functions of the optic nerve, like those of the posterior columns, are afferent; both optic nerves and posterior columns are liable to degenerative diseases of the same character; they are often affected together, or successively after a comparatively short time; they may in rare instances be independently attacked, as in some cases of so-called tabetic atrophy; and, finally, the visual apparatus can be made to substitute functionally the apparatus for general sensibility. One anatomical difference must, however, not be overlooked. The spinal process of the ganglion cell comes from the same cell as the peripheral process, and is not part of an independent nerve cell; it corresponds to the axis cylinder, and the peripheral process to the dendritic process. The connection of the retina with the central nervous system is never lost, as is also the case with the olfactory bulb; but the optic nerve differs from the olfactory nerve in that it is never associated by nervous elements with modified epithelial cells at the surface ectoderm. It is, therefore, the only portion of the central nervous system upon which external influences act directly, unless we assume that the lens and the surface epithelium of the cornea play a part in relation to the retinal elements comparable to that performed by the terminal nerve elements of the skin. The retina has been compared to a modified basal ganglion, and the optic nerves to association bundles bringing it into relationship with other parts of the brain. (Monro, Robinson, and Van Gehuchten.)

End Sheathing of the Optic Nerve.—Externally the optic nerve is supplied with a strong dural sheath, which becomes the sclerotic coat of the eyeball. It is closely invested with a pial sheath. Between the two is a delicate arachnoidean layer, which peripherally unites with the dural sheath, and hence the subdural and subarachnoid spaces become continuous as the eye is approached. The optic nerve is narrower at the lamina cribrosa, which is the scleral layer

transformed into a very open network to allow the passage of the nerve fibres. The separate fibres of the optic nerve are estimated as numbering four hundred and fifty thousand.

Structure of the Retina.—The retina is a delicate and highly evolved structure, and has been variously subdivided into layers. The researches of Ramón y Cajal, Van Gehuchten, Edinger, and others seem to show that it is best from the histological point of view to recognize three principal layers, namely, (1) the *layer of visual cells;* (2) the *layer of bipolar cells;* and (3) the *layer of ganglionic cells.* These three layers can be divided into other less well defined layers or strata. The layer of visual cells is the outermost of the retinal layers.

FIG. 353.

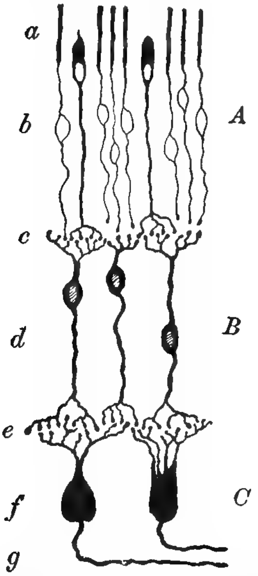


Cross-section of healthy optic nerve about five millimetres behind the eyeball (magnified eighteen diameters): *du*, dural sheath; *p*, pial sheath; *s*, subdural space; between the dural and pial sheaths are seen fibres of the arachnoid; *vr*, central vessels. (Wilder.)

The most distal portion of the peripheral prolongations (protoplasmic or dendritic processes) of its cells constitutes the *rods and cones* of the retina. The central prolongations of these cells pass into the deeper portions of the retina, where they end free. Beneath the layer of visual cells is the layer of bipolar cells. Its peripheral (protoplasmic or dendritic processes) terminate in arborizations which interlace and are in contact with the similar terminations of the central processes of the cells of the visual layer (forming the *external molecular layer*), while the axis cylinders or central prolongations of the bipolar cells end in their turn in an elaborate arborization in the depths of the retina. The deepest of the three great layers of the retina, the layer of ganglionic cells, is composed of cells of very large size, whose peripheral

(protoplasmic or dendritic) processes, terminating in a complex arborization, intermingle with the central arborizations of the cells of the bipolar layer (forming the *internal molecular layer*). Centrally each ganglionic cell has a single axis cylinder which becomes one of the fibres or elements of the optic nerve trunk. These fibres taken together at their origin are sometimes spoken of as the layer of nerve fibres. It will be seen that the subordinate layers or strata are the results in some instances simply of subdivision of the three layers, and in others of combinations of portions of two of these layers. Three of these subordinate layers not yet described are the *external granular layer*, the *internal granular layer*, and the *layer of ganglionic cells*. In the illustration (Fig. 354) are shown the main elements of the retina and its arrangement into three principal layers and other subordinate layers or strata. According to this description, three superimposed systems of nerve cells or neurons are found in the retina. The visual cells first receiving the luminous impressions, these are conveyed centrally to the bipolar cells, and from the latter by way of the axis cylinder processes of these ganglionic cells into the optic nerve trunk. The optic nerve fibres conduct these impressions to the primary basal centres in the interbrain and midbrain, and from these they are conveyed to the cerebral cortex. Besides the nervous elements just described, among these elements are found cells of an epithelial nature, which are usually regarded as sustentacular. In the retina also are certain cell bodies and their processes, which take a more or less horizontal direction, some of which seem to connect visual and bipolar cells which are separated by a considerable distance. Throughout the thickness of the external molecular layer are found cells of large size, called *spongioblasts*, with apparently no axis cylinders, but with numerous dendritic processes which are directed internally. Their functions have not yet been determined. Because of the absence, or apparent absence, of axis cylinders, Ramón y Cajal has suggested the name *amacrine cells* for the spongioblasts. The arrangement of the rods and cones varies according to the region of

FIG. 354.

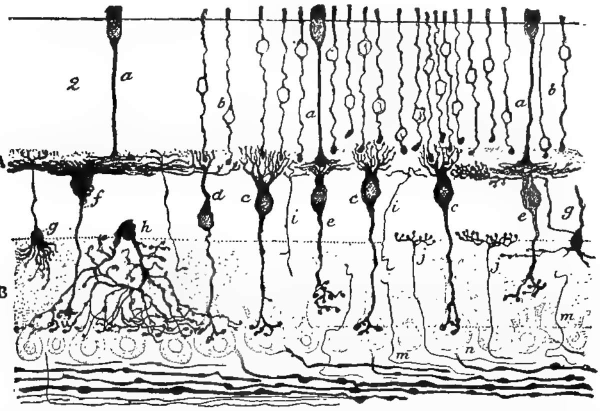


Layers of the retina: *A*, layer of visual cells; *B*, layer of bipolar cells; *C*, layer of ganglion cells; *a*, rods and cones; *b*, external granular layer; *c*, external molecular layer; *d*, internal granular layer; *e*, internal molecular layer; *f*, ganglion layer; *g*, layer of nerve fibres. (After Van Gehuchten.)

the retina in which they are found. In the macula the cones are closely packed together, being separated only by a single layer of rods, while in other regions they may be separated by three or four such layers. The cones are probably necessary for higher acuity of vision. According to Salzer, the cones of the human retina number about three million three hundred and sixty thousand, and Krause has estimated the rods at one hundred and thirty millions more. Further researches may render necessary some change in these estimates.

General Course and Connections of the Optic Nerves and Tracts.—Arising in the deepest of the retinal layers, the optic nerves traverse the choroid and sclerotic coats, the orbital cavities, and the optic foramina, reaching the base of the brain just cephalad of the

FIG. 355.



Section of the retina of a dog: *a, a, a,* cone fibres; *b, b,* rod granules and fibres; *c, c, c, c,* bipolar nerve cells whose erect distal arborizations pass to the central prolongations of the rods; *e, e,* bipolar nerve cells with horizontal arborizations which pass to the central prolongations of the cones; *f,* giant bipolar cell with horizontal ramifications; *g, g,* special elements with uncertain relations; *h,* diffuse amacrine cell (spongioblast); *i, i,* ascending axis cylinder processes; *j, j,* centrifugal nerve fibres; *m, m,* nerve fibres penetrating the internal molecular or inner plexiform layer; *n,* one of the ganglion cells receiving ramifications from the rod bipolar cells; A, external molecular layer; B, internal molecular layer. (Ramón y Cajal.)

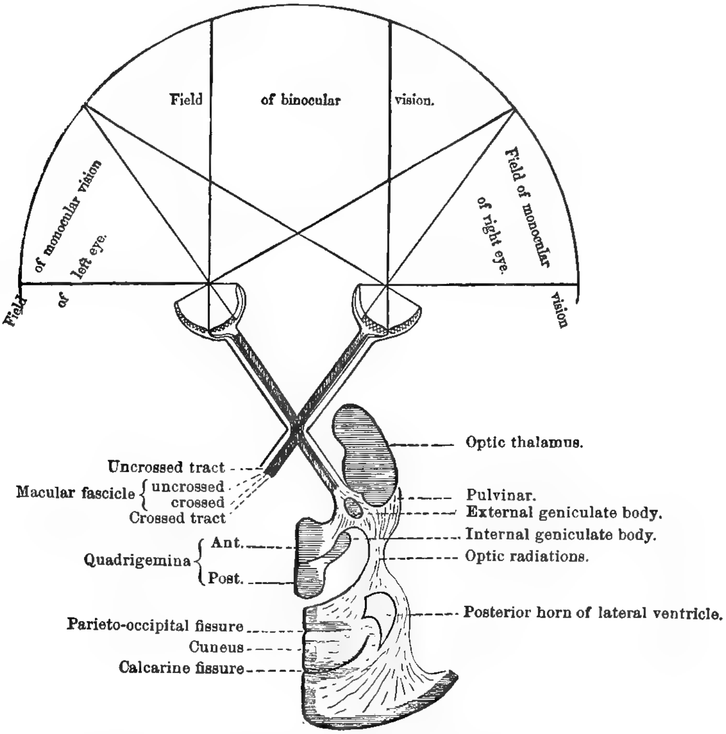
infundibulum. They here undergo partial decussation, forming the chiasm, which rests in the transverse furrow, called the optic groove, on the superior face of the sphenoid bone. Caudad of the chiasm the optic nerves become the *optic tracts* or *striæ.* Each optic tract as it passes backward winds around the cerebral crus on its way to the interbrain and midbrain. In most descriptive anatomies the fibres of the optic tract are spoken of as dividing into an *external* or *lateral* and an *internal* or *mesal* root. What is designated as the external root is in reality, however, the true continuation of the optic nerve and tract. The fibres of the so-called internal root are in the main derived from Gudden's and Meynert's commissures, and they do not in a proper sense form a part of the optic nerve and optic tract, although for a part of their course they are in imme-

diate juxtaposition with the latter. All authorities are not in accord as to the exact basal termini of the optic tracts. Usually it is held that the vast majority of the optic fibres terminate by free ramifications in the pregeniculum, the pregeminum, or the pulvinar. The visual centres in the interbrain and midbrain are connected with the calcarine cortex by a special band of cerebral fibres, the optic radiations of Gratiolet. The angular and the external occipital cortex are also connected with these basal centres indirectly through the calcarine cortex, and, not improbably, also by some direct bundles, although the passage of projection fibres directly to this region is generally denied. According to some authorities, a few of the fibres of the optic tract do not terminate in either the interbrain or the midbrain, but pass into the white substance of the cerebrum, eventually to reach the gray matter of the visual cortex of the same side. The evidence in favor of the existence of this tract is not, however, of a positive character, or at least is not conclusive. The optic tract is firmly attached by brain substance to the precribrum and the crus. The basal ganglia overlie the tracts. It is probable that the optic nerve has direct connection with the lenticula. Through the external capsule it is probably connected also with the first and second temporal convolutions.

Separate Fasciculi of the Optic Nerves and Tracts.—The fibres constituting the optic nerves and tracts are arranged into several separate bundles or fasciculi, each of which has a particular function. The main bundles, three great afferent visual fasciculi, are (1) a bundle of direct fibres which comes from the external or temporal third or fourth of each retina and passes into the optic tract of the same side; (2) a bundle of fibres which comes from the internal or nasal two-thirds or three-fourths of the retina and passes at the chiasm into the optic tract of the opposite side; and (3) a bundle of fibres which proceeds from the region of the macula and also decussates, passing partly to the optic tract of the same side and partly to the optic tract of the opposite side. The crossed fibres of this macular bundle probably come from the nasal side of the macula, and the uncrossed fibres from its temporal side. A scheme of the course and connection of the visual tracts from the retina to the cortex, and of the separate fasciculi of the optic tracts, is shown in Fig. 356. The first two fasciculi of the optic tract convey luminous impressions from all portions of the retina except the macula, but they do this, as just indicated, in unequal proportion. By means of their fibres those luminous impressions which are concerned with the general recognition of form and color are transmitted. The fasciculus of uncrossed fibres occupies the outer or lateral part of the chiasm, while that of crossed fibres makes up the more central larger part which is left. The macular bundle is that which brings the retinal area of clear vision into relation with special encephalic centres, basal and cortical. It

occupies about one fourth of the extent of the nerve, and is triangular. At first it is situated in the inferotemporal portion of the nerve, but as the nerve passes backward through the orbit this fasciculus gets near the centre, and just in front of the chiasm it occupies the dorsomesal portion of the nerve. In the optic tract it again passes into the central portion of the nerve, where it remains

FIG. 356.



Scheme of the general course and relations and separate fasciculi of the optic nerves and tracts, showing also the fields of monocular and binocular vision. (Hill.)

almost completely encased in the other bundles until the brain is reached. In the diagram Fig. 357 are shown the relative positions of the macular and other bundles at different points of the optic nerve and optic tract.

Fibres of the Optic Nerve of Central Origin.—It is now well known that the optic nerve and its tract are not composed entirely of fibres which arise in the retina; some have been shown to originate in the basal centres, their cells of origin being in the pulvinar, the pregeniculum, or the pregeminum. The functions of these peripherally directed optic fibres have not yet been satisfactorily established, but Ramón y Cajal supposes that the purpose of some of them is to act upon the protoplasmic processes of the amacrine cells or spongioblasts found in the internal molecular layer. The layers of

the retina to which they are distributed are rich in bloodvessels. A number of investigators have demonstrated the influence of the sympathetic nerve in the neck on the vessels of the retina, and the researches of M. Elinson have shown that a large number of cen-

FIG. 357.

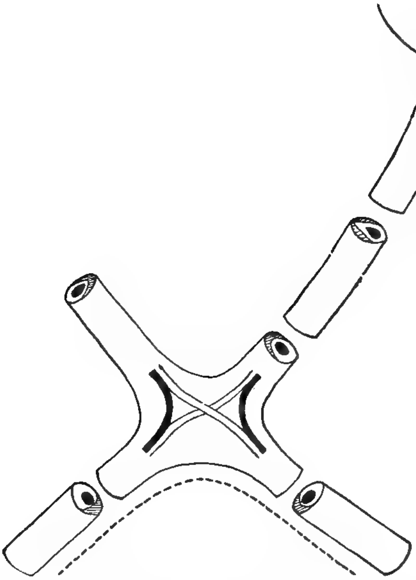


Diagram of the optic nerve and tract, showing the relative positions of the macular and other bundles in transections at different points. The position of the macular bundle, indicated in black, is in accordance with the views of Samelsohn, and that of the uncrossed bundles, shown by oblique shading, is according to Wernicke and Schmidt-Rimpler. The decussation of the macular bundle at the chiasm is shown, and the position of the commissural fasciculi posterior to the chiasm is indicated by the dotted line.

trifugal fibres of the optic nerve which he has traced into the retina have their origin in the sympathetic. It is probable, therefore, that a large portion of the fibres described by Ramón y Cajal as being in relation with the amacrine cells of the retina are especially destined for the innervation of its bloodvessels and do not play any direct rôle in the act of vision.

Partial and Total Decussation of the Optic Tracts.—Partial decussation of the optic nerves and tracts is the rule in man. In fish and birds this decussation is total. As an anomaly total decussation may sometimes be present in the human being. As the theory of total decussation seems to have recently received some support from high authority, it may be well to summarize briefly a few important facts bearing upon this subject. Jacobsohn extirpated one eye in rabbits, guinea pigs, cats, and monkeys, and, studying recent degeneration after the method of Marchi, found that in rabbits and guinea

pigs not a single degenerated nerve fibre could be traced through the chiasm to the optic tract of the same side, but that all such fibres passed to the tract of the opposite side. In the cat and in the monkey a large number of degenerated fibres passed to the optic tract of the opposite side, and also very many to the tract of the same side, showing that the decussation is partial in these animals. Studies in degeneration have also shown that in man the decussation is partial. Bernheimer reported in 1896 a case of old complete atrophy of the right optic nerve with a perfectly normal condition of the left nerve. The left optic tract contained a certain number of atrophic fibres, although not so many as the right tract. As the left optic nerve was perfectly normal, these degenerated fibres in the left tract must have come from the right optic nerve. This case is in accord with a previous observation of Bernheimer, and proves the presence of uncrossed fibres in man. Recent histological observations are not, however, in accord. Michel, for instance, asserts that he found a total decussation in the rabbit, dog, cat, and man. Ramón y Cajal found in the rabbit and mouse a direct optic bundle. According to Munk, the decussation is not complete even in birds. Koelliker found in man, and in the dog, cat, and rabbit, total decussation in every instance. Pick has confirmed the observations of Koelliker with regard to total decussation in the rabbit. We must, however, agree with the stand taken by Obersteiner, who, in reviewing Koelliker's work, asserts that total decussation of the optic fibres in man can hardly ever again be accepted by clinicians, in spite of the fact that so important an authority as Koelliker is in favor of this view. As suggested by Van Gehuchten, if total decussation at the chiasm is accepted, the well known cases of homonymous lateral hemianopsia from unilateral lesion of the occipital cortex can be explained only on the supposition of the partial decussation of the optic fibres which connect the basal centres for vision with the cerebral cortex.

Commissural Fasciculi (Gudden's commissure and Meynert's commissure).—In both Gudden's commissure and Meynert's commissure are probably situated the fasciculi which take part in the accommodation and the light reflexes. Gudden's commissure is the more important of the two bundles. It is situated in the caudodorsal portion of the chiasm, and constitutes about one third of its entire mass. It is sometimes difficult to separate the fibres of this commissure from those of the adjacent visual fibres proper. Meynert's bundle is situated in the dorsal portion of the chiasm, and is of much smaller size than Gudden's commissure. The fibres of this commissure, arising from the gray matter of the tuber cinereum, cross the middle line together, enter the crusta of the opposite side, and thence probably pass into the subthalamic body.

The Basal Centres for Vision and the Optic Reflexes.—The shrinkage of the pregeniculum after extirpation of the eye has been

clearly demonstrated. It has also been shown to undergo processes of atrophy or involution after removal of the occipital lobe or after a large lesion in this region of the cerebrum. According to Von Monakow, while the ground substance of the pregeniculum tends to disappear after removal of the eyeball, its cells remain. The end brushes of certain retinal cells terminate in the pregeniculum, and other large cells connected with vision here originate, the latter representing neurons which pass to the cortex. I do not know of any facts which prove that the macular fibres *alone* terminate in the pregeniculum, as Knies suggests. The sheet of white fibres which covers the thalamus and is known as the *stratum zonale* is composed, at least in part, of fibres of the optic tracts which pass over the pregeniculum, but just what course these fibres finally take is uncertain. The pulvinar, like the pregeniculum, is said to atrophy after destruction of the eyeball and the optic nerve. Histological research tends to show that, as in the case of the pregeniculum, fibres from retinal cells terminate in this portion of the thalamus, while cell bodies are found whose processes pass to the occipital cortex. It is not necessary to conclude, however, that these are true *visual* fibres, or that the nerve cells from the pulvinar to the cortex are visual in the sense that they convey impressions of light and color. The exact part played by the pulvinar in the mechanism of vision seems to be less well established than the functions of the pregeniculum and pregeminum, although the pulvinar is certainly an important organ intercalated in the visual pathway. Henschen believes that the fibres of the optic nerve which pass into the pulvinar are not visual, and this is the view that the author has taken in preceding pages (pp. 355 and 358). Degeneration of the pulvinar does not produce hemianopsia when the pregeniculum and optic radiations are intact, and we may have a lesion involving a large portion of the pulvinar without hemianopsia. When hemianopsia has been observed in cases of lesion of the pulvinar, the disease may not have been strictly confined to this ganglion. Both pulvinar and pregeminum are probably great reflex optic centres, but the problem of the functions of the former cannot be regarded as settled. Certain afferent fasciculi of the optic nerve pass to the pregeminum, in the nuclei of which are cells of large size. Axis cylinder fibres pass from these cell bodies, and in front of the nuclei of the third nerve decussate in the raphe. They become constituent fibres of the dorsal longitudinal bundle, and give off collaterals which ramify among the root fibres of the third, fourth, and sixth nerves, and thus is established a communication between the optic nerve fibres and the cell nests of all the motor nerves of the muscles of the eye. Lesions of the pregeniculum undoubtedly cause motor troubles of the eye and disorders of pupillary innervation. Pathological observations of recent date show that the pregeminum may degenerate without any essential change in vision. The affec-

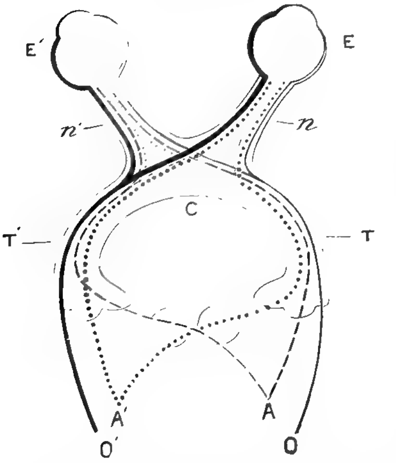
tions of vision referred to as occurring in connection with lesions of the pregeminum are of a reflex character, rather than true disorders of vision. The luminous impressions which invoke ocular and pupillary movements may be interfered with, but not those which awaken the cerebral sensations of light and color.

The Optic Radiations.—In the white substance of the occipital lobe are found large numbers of projection fibres which connect the basal centres for vision and for ocular and pupillary movements with the cortical visual region. These taken together are usually spoken of as the optic radiations of Gratiolet; but Gratiolet described only a portion of these fibres, those which he believed were directly traceable into the optic tracts. Certain projection fibres are also found here, which unite the cerebral cortex to the cortex of the cerebellum; still other fibres unite the occipital cortex with the most caudal portion of the internal capsule. Besides these projection fibres the subcortex of the occipital lobe contains a large number of association tracts which are in some way connected with visualization. Among these are tracts connecting the occipital with the temporal lobe, one convolution of the occipital lobe with other convolutions of the same lobe, and the occipital lobe of one side with the occipital lobe of the other by way of the splenium of the callosum.

Cortical Termini of the Visual Tracts.—Under visual localization (pp. 342–344) the subject of the cortical centres for vision has already been somewhat fully considered. It will be necessary here to recall that the primary cortical centres for vision are situated in the region of the calcarine cortex of both hemispheres, and the secondary or higher visual centres, including the centre for word seeing, in the angular and external occipital convolutions. As indicated under visual localization and in the discussion of aphasia (see diagram and legend, p. 629, Fig. 332), tracts proceed from the primary visual centres of the calcarine cortex on both sides to the higher visual centre of each side. The pure word blindness of Dejerine is caused by a lesion destroying the connection between both occipital lobes and the higher angulo-occipital region of the left side (see p. 634). Henschen regards the macula lutea as represented in the front of the floor of the calcarine fissure. Others hold that the macular region of the field of vision, if not represented in the calcarine fissure, has its representation in its immediate neighborhood. According to Ferrier, each angular gyre is in relation with the macula of the opposite side, partly by fibres which are supposed to decussate in the chiasm, and partly by fibres which decussate in the basal centres. The views of Ferrier as to macular representation are indicated in Fig. 358. The acceptance of the view that the macula has its representation in the angular and adjoining occipital region does not make it necessary to accept also the hypothesis as to a second decussation of the optic pathway in or behind the basal centres, although it is difficult to explain some of

the phenomena of central amblyopia except on this supposition. The macula, like all other portions of the retina, probably primarily has relations with the region of the calcarine cortex and secondarily with the angular and external occipital region. The macular bundle with its cortical continuation may preserve its separate place not only to the pregeniculum, but also through the optic radiations of Gratiolet to the primary cortical centres, and from these centres to the angulo-occipital region. The higher visual centre has its chief development in the left hemisphere. It is difficult to explain the cases of hemianopsia, single and double, in which a small area of clear vision remains, except on the supposition that the area of macular representation is in part at least in a portion of the cortex somewhat removed from the calcarine fissure. According to Von Monakow, the *exact* cortical area employed in seeing, and the exact relations of the primary optic centres to the visual cortex, have not yet been determined. He believes that the cortical area certainly includes more than the cuneus, lingual lobe, and descending occipital gyrus. He found that fibres connect the posterior part of the thalamus with the parietal lobe, and he believes that it is probable that a close connection exists between the angular gyrus and the superior parietal convolution on the one hand and the posterior part of the thalamus on the other, a small portion of the posterior part of the thalamus not being included in this connection. Von Monakow believes that the first, second, and third occipital convolutions are certainly included in the cortical visual area, and most probably also the superior and inferior parietal lobules. He holds that every portion of the external geniculate body receives fibres from the macula, and that these fibres are so projected cortically that they extend to the farthest limits of the visual cortex: so that when a lesion leaves only a small portion of the visual cortex intact, the fibres of this intact portion are sufficient for vision through the macula. Dejerine is, in part at least, in accord with those who hold that the angular convolution is a

FIG. 358.



Scheme of the optic tracts and visual centres: A, the right and A′, the left angular gyrus; C, optic chiasm; E, the right and E′, the left eye; n, the right and n′, the left optic nerve; O, the right and O′, the left occipital lobe; T, the right and T′, the left optic tract; the thin continuous line represents the retinal relations of O; the thick continuous line represents the retinal relations of O′; the interrupted line indicates the retinal relations of A, and the dotted line the retinal relations of A′; the relations of A and A′ with the eye on the same side are indicated by finer interrupted and dotted lines respectively. (Ferrier.)

higher visual centre. He believes that letters, like objects, are seen with the help of the visual centres of the calcarine cortex, but they are seen here simply like other objects, or like letters of an unknown tongue. In order that an assemblage of letters may awaken the idea of words, he holds that it is necessary that this cortical centre of common vision should enter into connection with the zone of speech, which is fully represented only in the left hemisphere, pathology showing that the centre of the visual memories of letters resides in the position of the angular gyre. Certain facts regarding the spectra and other visual sensations which occur in epilepsy and migraine seem, as suggested by Gowers, to postulate the existence of separate and higher centres. Gowers adopts the view of Ferrier, which has been subscribed to by the writer. Some of the most carefully recorded observations regarding the spectra of migraine show that these, whatever form they take, tend to occupy only the peripheral portion of the retinal field. The central field is nearly always free from these spectra, and the zigzag or other lines diminish in length as they approach it; but this central field is subject, on the other hand, to inhibition, becoming obscured or absolutely darkened.

Focal Lesions of the Visual Pathway.—*Definitions and Special Visual Symptoms.* Many of the terms used in the description of disorders of the visual apparatus have already been defined in whole or in part, and the methods of investigating disorders of this apparatus which especially interest the neurologist have been briefly described in Chapter II. Although the forms of hemianopsia are not described in detail, their general characteristics are there indicated by descriptive terms, and the exact significance of the special forms will appear when the lesions causing them are described. Hemianopsia of whatever type may be *partial* or *complete*—a part of the half field or the entire half field may be obscured. Hemianopsia may be *absolute*—the perception of light, form, and color may be wanting; or it may be *relative*—one or two of the special functions of sight being lost, while the others may be retained. *Double hemianopsia* is a very unusual form of visual disorder, in which, as the result of successive or simultaneous attacks of homonymous lateral hemianopsia, the entire field of vision, with the exception of the central or macular area, is obscured (see pp. 343, 344). Great irregularities in the method of obscuration of the visual field and in its degree may be met with, particularly in connection with disseminated lesions. It is rare to find hemianopsia present as an absolutely isolated symptom. For total loss of vision *blindness* is the simplest and best term. Formerly the word *amaurosis* was much used in the description of loss or impairment of vision, but it is being displaced by terms more exact and special. *Amblyopia* is most commonly used to designate partial loss of vision from various causes.

Color Blindness. The color sense is the faculty by which colors

are distinguished. *Achromatopsia* is color blindness. It is total only in extremely rare cases, and even when total the individual may distinguish differences in brightness or luminosity in what are to others different colors. When the loss for color is in one half field the affection of sight is designated *hemiachromatopsia*. *Dyschromatopsia* is difficulty in distinguishing colors, and the term *parachromatism* is sometimes applied to a false or incorrect perception of color. Hemiachromatopsia from a focal lesion is rare, but in one case observed by de Schweinitz the patient at first was absolutely hemianopsic, although later the form sense and the light sense returned, the hemiachromatopsia remaining. The case was one of homonymous lateral hemiachromatopsia. A few other cases have been recorded. Partial color blindness is usually subdivided into *red-blindness*, *green-blindness*, and *violet-blindness*. Dalton suffered from red-blindness: hence color blindness is often called *Daltonism*. It is doubtless true that in different cases different portions of the visual apparatus may be affected. The normal retina appreciates the colors in different degrees in its different parts. In the extreme peripheral portion of the field of vision colors are not recognized, everything appearing gray; as the periphery of the field is left, the difference between blue and yellow becomes appreciable; and still nearer to the middle, red and green are differentiated. The congenital hereditary forms of color blindness are, as a rule, due to an arrest of cerebral development, the defect not being in the perceptive but in the apperceptive portion of the visual apparatus. According to some authorities, the portions of the cortex which respond to different color impressions are distinct, although they may be near to each other; according to others, the different powers of color apperception reside in different layers of the cortex; but the exact truth remains to be discovered. Certain disturbances or lesions of the conducting fibres of the optic tracts give rise to optic disturbances in color vision. In some of these cases the visible spectrum gradually narrows from both sides, red, orange, and yellow on the one side, and violet and blue on the other, becoming less and less distinct, and thus, step by step, the entire middle portion of the spectrum becomes colorless. (Knies.) According to one largely accepted view, the defect in some cases of color blindness is in the retina, and the mechanism of its occurrence is variously explained. The three main colors which include all color sensations are supposed to be dependent upon the stimulation of three sets of retinal fibres, and this or that form of color blindness is present according to the particular retinal fibres which are affected. Another theory is that color blindness is due to the absence of some substance in the retina, red-green blindness being due to the absence of the red-green substance, and other forms to the absence of other substances. Cases of uniocular color blindness would certainly seem to show that, in some instances at least, the dis-

ease or defect producing the color blindness must lie in front of the chiasm, and, as in these cases visual acuity has not been reduced, the disturbance has most probably been in the retina. The same nerve may be the conducting apparatus for different colors, the brain being the differentiating organ, the nerve fibres simply undergoing different changes according to the colors that strike the nerve termini; or the retina is the differentiating or sifting organ, and the nerve tract conveys the color stimuli, already differentiated, to the special centres in the brain. The discussion of the entire subject of color blindness is foreign to a work of this kind. The chief interest of the neurologist is centred on those cases of this defect which are due to cerebral disease.

Forms of Central Amblyopia. A lesion involving any portion of the visual apparatus from the tips of the rods and cones to and including the cortex of the higher visual centres gives rise to affections of sight. Macular or central amblyopia may be produced by destruction of the macula itself, but lesions of intraocular origin come almost exclusively under the attention of the ophthalmologist. Central amblyopia will result from a small infiltrating lesion, a nodule of sclerosis or a focus of hemorrhage which may, by rare chance, be so situated as to destroy the macular bundle alone in any part of its course. Clinical experience has also shown that this bundle is especially liable to be affected by certain toxic agents (see Toxic Amblyopias). As the fibres of the macular bundle are undoubtedly distributed to the pregeniculum, a lesion of this body or of a special portion of it might lead to central amblyopia. While it is probable, it cannot, however, be regarded as fully settled that the pregeniculum is the basal centre for all portions of the retina. Knies, who believes that the pregeniculum is the special basal terminus of the macular bundle, has suggested that lesion of this body on one side should cause a partial amblyopia in both opposite halves of the fields of vision, and if both pregeniculi are destroyed, central vision should completely disappear. A lesion of any of the fasciculi of the occipital lobe which constitute the continuation of the macular pathway from the pregeniculum to the cortex will probably cause central amblyopia, partial or complete. A lesion of either the primary or the secondary cortical centre for the macula should also cause central amblyopia, although some facts would seem to show that this results from cortical lesions only when they are situated in or near the angular convolution. Lesions of the left cerebral hemisphere produce more positive effects upon central vision than those of the right. It is probable that partial central amblyopia, including pure word blindness, is caused by a lesion in the tract or tracts which pass from the primary cortical optic centres of the calcarine cortex of both sides to the angular region of the left side. The term *crossed amblyopia* has been used by Charcot, Ferrier, and Gowers to describe an affection in

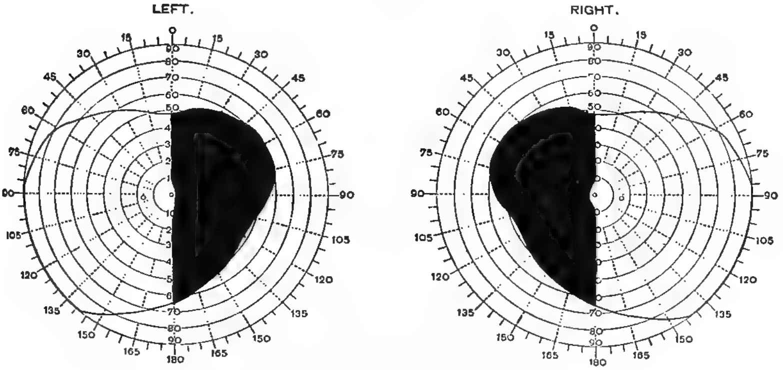
which the patient suffers from obscuration of vision in the eye opposite to the side of the cerebral lesion. A few clinical cases are on record in which a lesion of the angular convolution and its immediate neighborhood has apparently caused this symptom. Pathological observations and some experimental facts seem to show that the field of the same side, as well as that of the opposite side, is affected as the result of such lesions, some amblyopia usually being present on the side of the lesion, but less in degree than on the opposite side. Occasionally a very remarkable condition results from limited visual disturbances, of which I have seen two illustrations. The patients have what might be termed macular or central hemianopsia, or hemianopsic amblyopia. One half of the central field is obscured or lost, the peripheral field being maintained or, it may be, partially obscured. The patient is half blind for words. A word of more than four or five letters will only be half seen, or at least will be partially cut off unless the patient turns his eyes so that the unimpaired half of the central field shall take in different parts of the word in succession. Such a visual defect may be due to destruction of a part of the convolutional area for word vision.

Symptoms of Focal Lesions in front of the Chiasm. As the optic nerve has a length of about five centimetres (1.97 inches) from the retina to the chiasm, it may be affected by a gross but small focal lesion in this position. It may, for instance, be compressed by a gliomatous growth, an orbital exostosis, a thickening of the dura, or a hemorrhage. A lesion affecting the nerve in this part of its course is most likely to be situated in the orbit, as about three fifths of its length is here located. The symptoms will depend upon the extent and destructiveness of the lesion. Total destruction of the nerve will of course give total blindness in the corresponding eye. In some cases, as when the nerve is gradually compressed, sight disappears step by step, concentric contraction of the field gradually taking place. An infiltrating or an irregular lesion may destroy only portions of the nerve, and give corresponding limitations in the field. In addition to the loss of vision, the pupillary and other reflexes which are completed through the nerve are abolished.

Symptoms of Lesions involving the Chiasm, including Forms of Heteronymous Hemianopsia. A destructive lesion involving the entire chiasm causes complete blindness. When a lesion involves the optic nerve in front of the chiasm, and also in part the chiasm itself, in addition to blindness in one eye the fields of vision in the other eye will be affected according to the degree of the chiasmal implication. *Nasal hemianopsias* result from lesions affecting the uncrossed fasciculi which are usually regarded as passing in the lateral portion of the nerve, chiasm, and tract from the external or temporal portion of the retina. A unilateral lesion sometimes compresses one side of the chiasm and gives rise to a *unilateral nasal*

hemianopsia. Binasal hemianopsia is rare, but cases have been reported in which it has been produced by calcified and enlarged carotid arteries pressing upon both sides of the chiasm. Irregular nasal hemianopsia has been reported as occurring at certain stages of toxic or tabetic cases, the explanation evidently being that in these cases the lateral fasciculi of the nerves and tracts are first attacked. When a lesion occurs so as to destroy the caudal or posterior portion of the chiasm, *bitemporal hemianopsia* results, to produce which the lesion must be so placed as to destroy only the crossed fibres. Bitem-

Fig. 359.



Visual fields in binasal hemianopsia. (Bramwell.)

poral hemianopsia has been recorded in acromegalia, and this may have been due to hypertrophy of the hypophysis, a lesion which has been observed in several instances of this disease. Other cases of bitemporal hemianopsia have been reported in connection with fractures, and in intracranial gummata and sarcomata. In a case reported by Weir Mitchell an aneurism of an anomalous artery connecting the two carotids was present, and had destroyed the chiasm in the median line, causing bitemporal hemianopsia. When a lesion at the chiasm is so placed as to destroy not only both sets of crossing fibres but also one set of lateral fibres, hemianopsia with complete loss of vision, on one side, will be present. Infiltrating or other forms of irregular lesion of the chiasm may give rise to combinations of different forms of partial hemianopsia with partial or complete central amblyopia. Lateral hemianopsia of one eye has been noted in association with obscuration of three quadrants of the other eye. A form of transient bitemporal hemianopsia, *hemianopsia bitemporalis fugax*, is sometimes observed in syphilitic subjects, and this may yield rapidly to the iodides or mercurial inunction. Oppenheim has recorded several such cases due either to a syphilitic new growth or to a gummatous meningitis. Not all such cases yield fully to specific treatment, necrotic processes sometimes having taken place. In Fig.

362 is shown a frontal section of a gumma overlying the chiasm. Under the general designation of *heteronymous hemianopsia* several of the irregular varieties of hemianopsia just described are usually classified, the term being used as contradistinctive to *homonymous.* Thus the different forms of nasal and temporal hemianopsia are sometimes spoken of as heteronymous. In the homonymous forms of this disorder the visual obscuration affects corresponding portions of the visual field, that is, portions of the visual field of each eye which ordinarily act together; while in the heteronymous hemianopsias portions of the visual field which do not ordinarily act in conjunction are obscured.

FIG. 360.

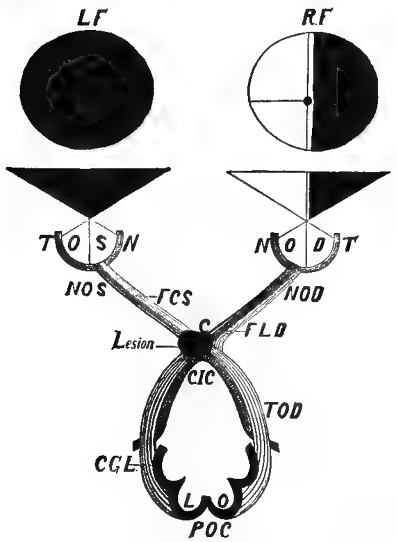


FIG. 361.

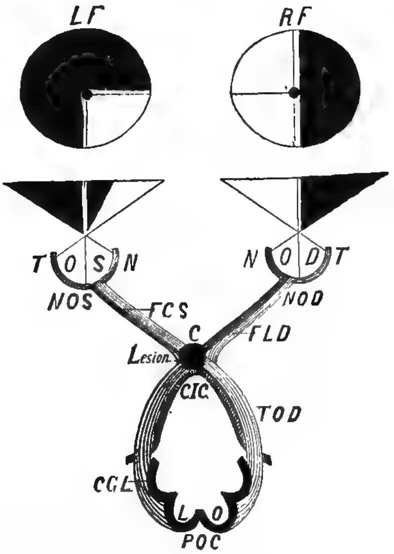


FIG. 360.—Chiasm hemianopsia: loss of vision in left eye, temporal hemianopsia in right eye; complete atrophy of the left optic nerve, partial atrophy of the right; no other symptoms of cerebral lesion; hemiopic pupillary inaction.

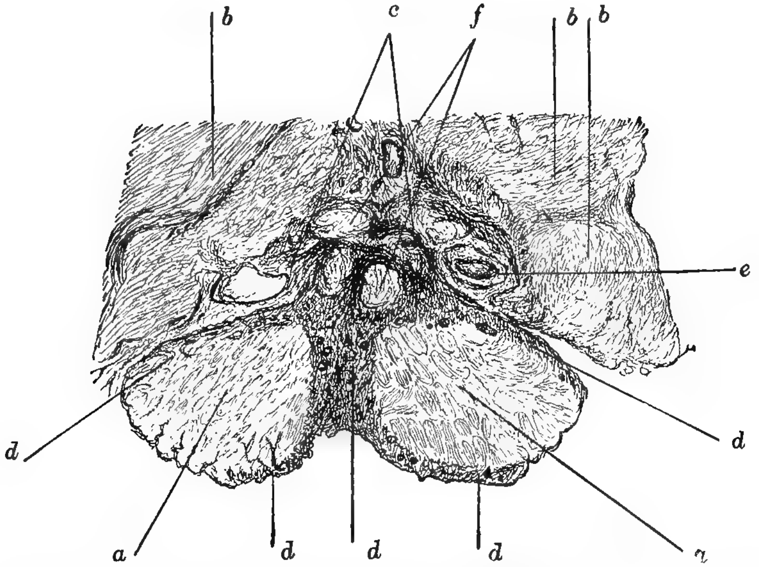
FIG. 361.—Chiasm hemianopsia: partial blindness, with marked atrophy of both optic nerves; bitemporal hemianopsia plus obscuration of upper nasal quadrant of left field; no other symptoms of cerebral disease; hemiopic pupillary inaction in right eye.

LF, left visual field; *RF*, right visual field; *OS*, left eye; *OD*, right eye; *N* and *T*, nasal and temporal halves of each retina; *NOS*, left optic nerve; *NOD*, right optic nerve; *FCS*, left crossed bundle; *FLD*, right uncrossed bundle; *C*, chiasm; *CIC*, commissural fasciculi (non-visual fibres of chiasm); *TOD*, right optic tract; *CGL*, pregeniculum, and *LO*, pregeminum, which together make up *POC*, the basal primary optic centres. (Seguin.)

In what has just been said about the forms of heteronymous hemianopsia from lesions involving the chiasm, the usually accepted views with regard to the position of the decussating and nondecussating tracts of the chiasm have been followed. These views are not, however, universally held, and observations of Wernicke and Schmidt-Rimpler would seem to show that the uncrossed fibres at the chiasm coalesce into a single bundle, which lies to the inside and a little inferiorly, instead of laterally as usually held. Schmidt-

Rimpler traced bands of degeneration which indicated this course in a case of hemianopsia. In the diagram Fig. 357, showing the position of the macular bundle, the course of the uncrossed fibres according to Schmidt-Rimpler is also shown by means of shading. As remarked by Knies, if we assume that the course of the fibres in the optic nerve and chiasm is like that described by Wernicke and Schmidt-Rimpler,

FIG. 362.



Frontal section of gumma overlying chiasm, four times magnified: *a*, *a*, optic nerves; *b*, *b*, brain substance; *c*, *c*, main mass of gumma; *d*, *d*, new growth extending into chiasm, between and around optic nerves; *e*, vessel with thickened and infiltrated walls; *f*, necrotic spots within gumma. (Oppenheim.)

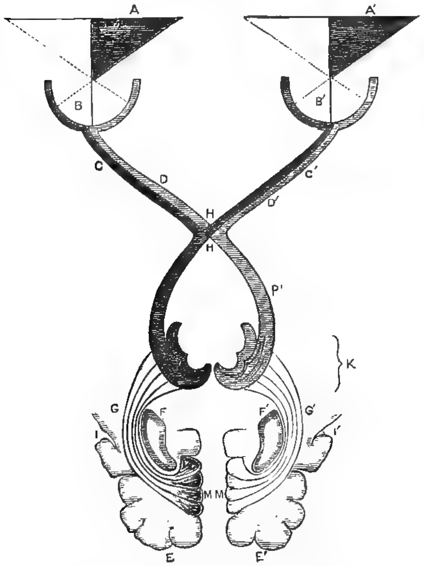
then a single lesion of the anterior angle of the chiasm between the two optic nerves might give rise to a condition similar to binasal hemianopsia. The probabilities are, however, that the course of the crossed and uncrossed bundles as described by Schmidt-Rimpler is exceptional.

Symptoms most frequently associated with Heteronymous Hemianopsia. Heteronymous hemianopsia, which, as just shown, usually points to a chiasmal lesion, is commonly associated with other symptoms of disease at the base of the brain, a knowledge of which will facilitate exact diagnosis. Binasal hemianopsia may be associated with paralysis of all or nearly all the motor and sensory nerves of both eyes, and this indicates a chiasmal lesion spread out so as to affect the orbital nerves, leaving the central decussating fibres of the chiasm intact. Bitemporal hemianopsia may be associated with unilateral or bilateral anosmia, indicating disease of the mediofrontal edges of the chiasm and extending forward. When bitemporal hemianopsia is

present with blindness of one eye (later) and paralysis of some or all of the nerves which enter the orbit of the blind eye, the lesion is probably laterad of the chiasm, involving the orbital nerves and the optic nerves as well as a large part of the chiasm. (Seguin.) The pupillary reflexes will be abolished in part in these cases. The light reaction cannot be obtained from the blind portion of the field. The accommodation reaction and the reaction to cutaneous irritation will be present. It cannot be emphasized too much that in cases of chiasmal lesion the visual defects may be of a very irregular character.

Homonymous Lateral Hemianopsia. In homonymous lateral hemianopsia, the most common form of hemiopic defect, the temporal field of one eye and the nasal field of the other are darkened to the right of the fixation point, or the corresponding fields to the left, causing the left or right form of homonymous lateral hemianopsia. This form of hemianopsia may be caused by lesions situated in the optic tract back of the chiasm, in the pregeniculum, in the optic radiations, or in the calcarine cortex. According to some, it may also be caused by lesions of the pulvinar. Forms of incomplete lateral hemianopsia are most frequently due to lesions of the occipital cortex or subcortex. In a considerable percentage of the cases of homonymous lateral hemianopsia, a small, central, semicircular area representing a portion of the macular field is not darkened, while in other cases the hemianopsia is complete. The exact position and extent of the lesions giving each of these two forms of hemianopsia have not yet been clearly demonstrated.

FIG. 363.

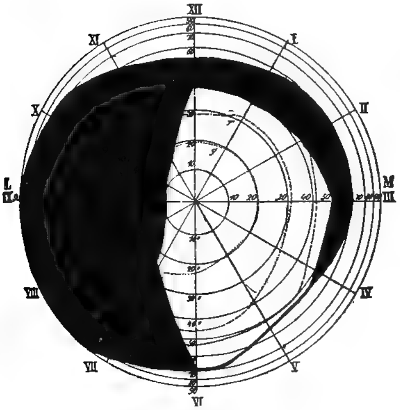


Right homonymous lateral hemianopsia, from lesion of the left visual centre of the cortex or left optic tract: A, dark left nasal half field from blind temporal half of retina; A′, dark right temporal half field from blind nasal half of retina; B, left eye: B′, right eye; C, C′, left and right optic nerves, composed of the crossed bundles of fibres; D, D′, left and right crossed bundles; E, E′, left and right occipital lobes; F, F′, left and right posterior horns; G, G′, optic radiations; H, H′, optic chiasm; I, I′, angular gyrus; K, region of optic thalamus, geniculate body, and quadrigeminal bodies, collectively termed the primary optic centres; M, M′, cuneus. The left cuneus and optic tract are shaded, to show lesion of these parts and the influence of the lesion upon the retina.

The Association of Hemianopsia with Hemiplegia. Hemiplegia, partial or complete, transient or permanent, is a frequent association of hemianopsia. In many cases of hemianopsia with hemianesthesia partial hemiplegia is also present. In these cases the lesion prob-

ably involves directly or indirectly the internal capsule, and when the hemiplegia is prominent and persistent the capsule may be involved almost the entire distance from its knee to its extreme caudal portion. Transient hemianopsia may be present in the early stages

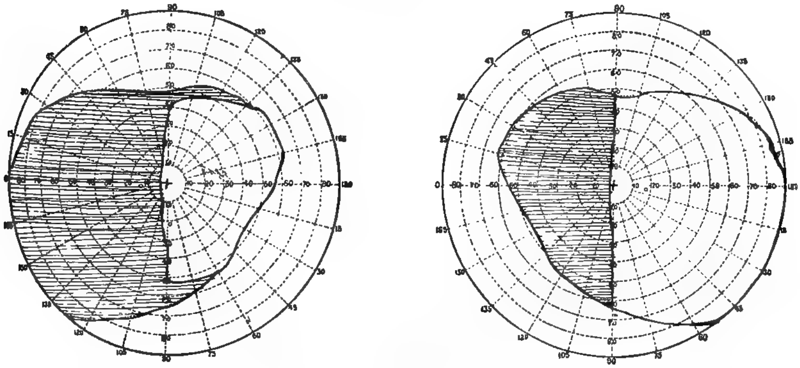
Fig. 364.



Visual fields in a case of left homonymous lateral hemianopsia. (Starr.)

of an apoplectic attack with hemiplegia and other unilateral manifestations, disappearing in a few hours or days. In these cases the hemianopsia is probably due to the effects of pressure or inhibition, or of both, upon the parts of the visual pathway which are not included in the destructive lesion. It is not well, therefore, in the early stages of some apoplectic cases to draw conclusions as to the

Fig. 365.



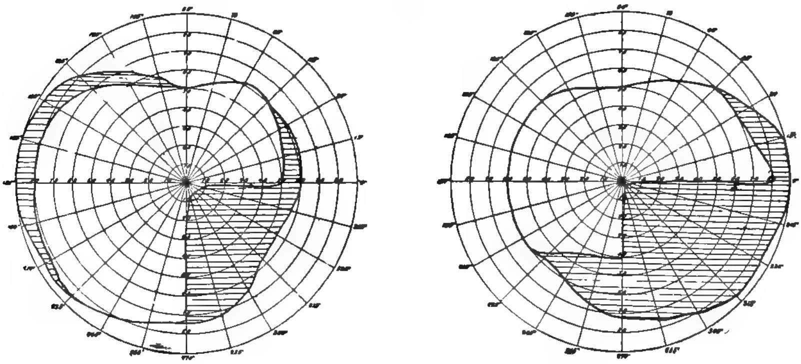
Visual fields in a case of left lateral hemianopsia.

exact extensions of a lesion based upon the presence of hemianopsia. In nearly all cases the loss of sensation and of power, and the other disorders of motion which accompany hemianopsia, are on the same side of the body as the blind half fields. Potts has reported a case

of hemiplegia of the right side with hemianopsia of the left, caused by a penetrating wound of the brain; but in this case evidently the optic nerve on one side and the motor region of the brain on the other were injured.

Partial and Special Forms of Hemianopsia. Various partial and special forms of hemianopsia are sometimes observed. The defect may be limited to a portion of the half field. Some interesting cases of special forms of quadrant or sector defect from cortical lesions have been reported. In a well-known case reported by Hun, for example, in which the lower lateral half of the visual field of one side was obscured, the lesion involved only the lower portion of the cuneus. The obscuration may extend only over a quadrant, a sector, or some irregular segment of the retina, and this may or may not be entirely confined to one side of the vertical line. The term hemianopsia, which etymologically refers to an impairment or loss of vision in a half field, is not strictly applicable to these defects, but it is sometimes used as a matter of convenience, generally with a qualifying expression indicating the limitation of the obscuration: thus, quadrant hemianopsia, sector hemianopsia, or segmental hemianopsia may be spoken of in discussing the visual disorder. Properly speak-

FIG. 366.

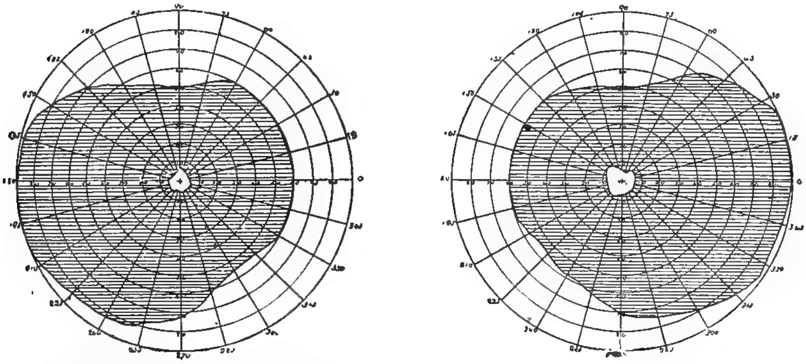


Visual fields in a case of right lateral quadrant anopsia.

ing, these visual defects are *anopsias.* When the darkening of the field of vision in these forms of partial hemianopsia is largely or altogether confined to one side of a vertical line passing through the fixation point, the lesion causing the visual defect is usually a limited one in some of the positions already spoken of, as in the optic tract, pregeniculum, optic radiations, or calcarine cortex. In a large majority of these partial cases, however, the lesions are situated in the occipital cortex or subcortex, and particularly in the former. When a quadrant or sectorial defect is due to limited lesion of the cortical visual zone, the symptom usually stands almost alone; but sometimes alexia, sensorial agraphia, or the so-called psychic blindness may be present.

When sectorial defects originate from lesion of the occipital subcortex, the defective portions of the fields are not likely to be so well defined in boundaries as when the lesions are cortical. The loss of light sense will probably be less complete than in cortical cases, and irregular spaces where light is recognized may be mingled with the blind spaces. Sectorial, quadrant, or other partial defects may arise from lesions of the optic tracts, but when this is the case they will be accompanied by other significant symptoms of basal disease, and, as in all cases of lesion of the optic tract, the senses of light, form, and color will be lost together. (Noyes.) The appearance of the fields in a case of double hemianopsia, caused by successive attacks of lateral hemianopsia, is shown in Figure 367.

FIG. 367.



Visual fields in a case of double hemiplegia with double hemianopsia and loss of orientation, the result of two successive apoplectic attacks: the small central white area represents the limits of the preserved field (macular vision). (Dunn.)

Horizontal Hemianopsias. Instead of the hemianopsia being lateral or vertical, it may be *horizontal* or *altitudinal*. In horizontal hemianopsia the line which divides the fields of obscured and preserved vision is horizontal. The obscuration may be either *superior* or *inferior*, in the former the upper half fields being darkened, and in the latter the lower half fields. The superior and inferior forms of horizontal hemianopsia, like vertical hemianopsia, may be partial; in other words, the obscuration may be limited to a smaller or a larger portion of the upper or lower fields. Forms of horizontal hemianopsia may be due to a lesion of the chiasm or optic tracts so situated as to destroy or compress either the dorsal or the ventral fibres of the tracts in these positions. They might also be due to a lesion in the eye, as, for instance, to embolism of the central artery of the retina, or to circumscribed retinitis; and it is also possible that symmetrical, bilateral, cortical lesions may involve such portions of the cortex on both sides as to cause a superior or an inferior form of hemianopsia. In Hun's case of lesion of the cuneus a part of the inferior fields was obscured.

Symptoms associated with Hemianopsia when the Lesions present involve the Basal Centres for Vision and the Optic Reflexes. While it is rare to have a lesion isolated to either of the so-called basal primary centres for vision, gross lesions involving one or more of these bodies and the regions lying adjacent to them are comparatively common, and hemianopsia has been noted in a number of the cases in which such lesions have been recorded. As indicated in the discussion of the relations of the pregeniculum to the visual pathway, it is probable that a lesion of this ganglion will cause hemianopsia of complete or incomplete type, and it is also probable that lesions of this ganglion or of a portion of it will cause central amblyopia. The hemianopsia in such cases can therefore properly be referred to the lesion of the pregeniculum itself, or it may be conjointly due to involvement of this basal centre and of the tracts which enter or leave it. Other symptoms likely to be present will be those of disease of the thalamus and possibly also of the quadrigeminum, as these ganglia lie so close together that they may be easily involved in a gross lesion. Oculomotor paralysis from lesion of the third nerve, and hemiplegia or hemiparesis, might result, if the lesion were of considerable size and extended into the crus. The hemianopsia in these cases is of the homonymous lateral type, and hemiopic pupillary inaction is present. Hemianopsia has certainly been observed in a considerable percentage of cases of disease of the thalamus, and this fact is used as an argument in favor of the view that the pulvinar is to be regarded as a true basal visual centre. Amblyopia is also sometimes present, but in other cases visual symptoms have been absent. Whatever view is taken, the clinician should be familiar with the symptoms which are commonly associated with hemianopsia in cases of thalamic disease. These are largely the same as those which accompany hemianopsia when the external geniculate body is implicated. The symptoms will, of course, vary according to the extent of the destruction of the thalamus. In several places in preceding pages the effects of lesions of different portions of the thalamus have been considered (pp. 355–358). Besides the visual symptoms, sensory, motor, auditory, olfactory, thermic, vasomotor, and psychic symptoms have been recorded. The symptoms most commonly associated with hemianopsia in thalamic lesions are hemianesthesia, hemiataxia, hemichorea, and hemiparesis or hemiplegia. The hemianopsia may in these cases be due to lesion of the pregeniculum or of the adjoining optic radiations. When the lesion is limited to the pregeminum, visual symptoms, strictly speaking, are not caused, but the proper coordination of visual impressions and pupillary and other ocular movements will be prevented. So far as conjoint movements of the eye are dependent upon visual impressions received by the eye opposite to the lesion, these will be abolished in lesions of one pregeminum. Disorders of station and locomotion, clonicotonic spastic phenomena,

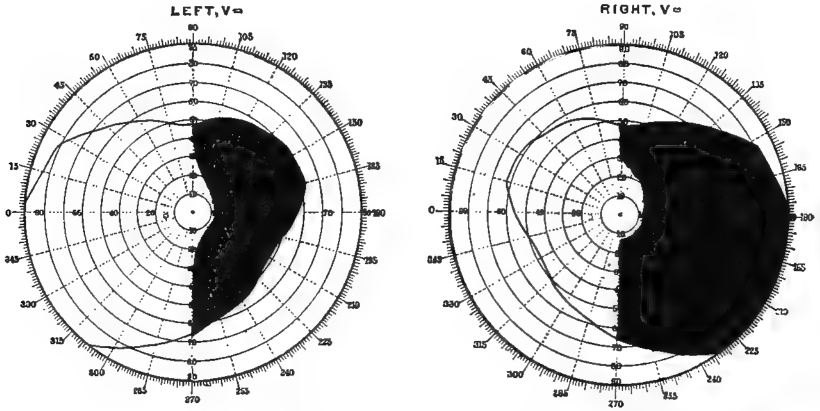
and other symptoms which have been considered under lesions of the quadrigeminal body may be present. (See pp. 370 and 371.) When gross lesions involve all the primary optic centres, as in some cases of tumor, the resulting symptoms will be of a most complex character; but a careful consideration of the phenomena to be expected from implication of each of these ganglia may enable the clinician to unravel the confused tangle which is presented to him for diagnosis. The hemiopic pupillary inaction will be present when any of these basal centres are the sites of the lesions. Knies has called attention to a peculiar and highly interesting symptom which might result from isolated lesion of the pregeminum. As the fibres which go to the pupillary sphincter pass close to or through the pregeminum, if these are destroyed on one side at the same time as the pregeminum, the hemiopic pupillary reaction (inaction) without hemianopsia would result, but the symptomatology might readily escape observation. When quadrigeminal disease is suspected, the patient should therefore be tested for Wernicke's hemiopic pupillary inaction.

Association of Hemianopsia with Disorders of Ocular Movements. When homonymous lateral hemianopsia is associated with some of the forms of alternate hemiplegia the lesion is most probably situated at the base of the brain in such a position as to injure the optic tracts and crus and adjoining structures. A combination sometimes presented is that of paralysis of the third and fourth, and more rarely of the fifth and sixth, cranial nerves, on the side of the lesion, with homonymous lateral hemianopsia, and hemiplegia or hemiparesis on the opposite side. In such a case the hemiopic pupillary inaction will be present, as in all cases in which the lesion is situated so as to involve the optic tracts or the basal primary optic centres.

Association of Hemianopsia with Disturbances of Speech. Various forms of disturbance of speech are somewhat frequently associated with hemianopsia. The position of the lesion in these cases is in large part to be determined by a study of the exact nature of the speech defect, which is sometimes a motor aphasia, at other times a sensorimotor aphasia; or it may be word blindness, dyslexia, verbal amnesia, or word deafness. According to Seguin, lateral hemianopsia with typical hemiplegia, spastic after a time, aphasia if the right side be paralyzed, and with little or no anesthesia, is certainly due to an extensive superficial lesion in the area supplied by the middle cerebral artery. Cases with lesions so extensive as this are rare, but when a true motor or sensorimotor aphasia occurs with hemianopsia, destruction of a considerable portion of the region supplied by the Sylvian artery should be expected, with also lesion of the occipital lobe. In the majority of cases of hemianopsia the associated speech disturbance is of the sensory or concept variety. Cortical word blindness or the pure word blindness of Dejerine may be present, the lesion in these cases in all probability involving the

parieto-occipital cortex or subcortex, especially on the left side. When in these cases, besides the lateral hemianopsia and the word blindness or dyslexia, there is also hemianesthesia, the lesion involves both the caudal portion of the internal capsule and the white matter of the parietal and occipital lobes. When hemianopsia is associated with verbal amnesia and partial word blindness, the lesion is probably situated in the inferior temporo-occipital region, as in one of my cases, in which the patient suffered from verbal amnesia, partial word blindness, and later from partial hemiplegia, with partial homonymous lateral hemianopsia, as shown in the diagram Fig. 368. The lesion in this case was found to be a tumor involving the midtemporal and temporo-occipital region. Word deafness may be associated with homonymous lateral hemianopsia, as in a case reported by Dr. Spiller and the writer, in which an abscess was situ-

Fig. 368.



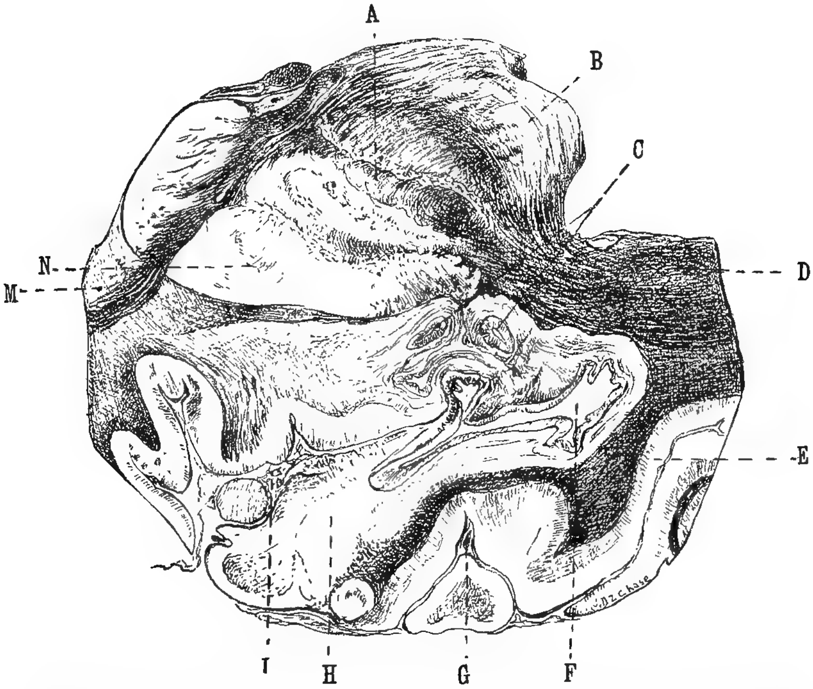
Visual fields in a case of verbal amnesia with lesion of the midtemporal and inferior temporo-occipital cortex and subcortex.

ated at the posterior part of the external capsule, involving the medullary substance of the first temporal convolution and also the posterior part of the lenticular nucleus. A microscopical section through the lesion in this case is shown in Fig. 369. This patient had had several epileptiform convulsions, was partially paralyzed on the right side, and could not talk properly, although the exact nature of his aphasia was not determined. The loss of function in the motor and visual tracts was probably due to pressure exerted by the pus in the abscess cavity, but the hemianopsia persisted during the time that the patient was under observation. The peculiar site of this lesion should be remembered in cases in which hemianopsia is associated with verbal deafness.

Association of Hemianopsia with Loss of the Color Sense. Hemiachromatopsia is a comparatively rare accompaniment of hemianopsia. When the perception of light is wanting, necessarily that of form

and that of color are also wanting; but if the light sense is preserved, either the color sense or the form sense or both may be deficient or absent. When the color sense (and the same is true of the form sense) is lacking or lost, the light sense being preserved, meagre clinical and pathological data show that the defects are most commonly, if not invariably, due to lesion, direct or indirect, of the visual cortex. This subject has been studied by Swanzy, Noyes, and others. At least one case has been recorded with an autopsy, by

Fig. 369.



Lesion in a case of homonymous lateral hemianopsia with word deafness; microscopic section (unmagnified) from the left hemisphere at the level of the parallel fissure; the first temporal gyrus extends to about three-eighths of an inch below the level of this section; the abscess cavity terminates a short distance below this level: A, posterior limb of internal capsule; B, thalamus; C, separated terminations of the abscess cavity; D, optic radiations; E, second temporal gyrus; F, chief portion of abscess cavity; G, parallel (supertemporal) fissure; H, degenerated first temporal gyrus; I, Sylvian fissure; M, anterior limb of internal capsule; N, putamen. (Mills and Spiller.)

Verrey. A lady sixty years old, after an apoplectic attack, had absolute color blindness in the right half of each field, with reduced but not complete loss of light sense and form sense in the same region, but without dyslexia and mental blindness. The lesion which had caused this defect was an old hemorrhagic cyst in the lower part of the left occipital lobe, extending into the temporal lobe on the mesal side. The cyst was situated between the floor of the posterior horn of the left lateral ventricle and the basal surface of the left occipital lobe.

It had occupied the white substance of the inferior occipital convolution, and had almost completely destroyed the white substance of the posterior extremity of the occipitotemporal convolutions, as well as that of the posteroinferior part of the cuneus. The cyst had destroyed the deeper layers of the cortex and of the cuneus and other convolutions mentioned. (Swanzy and Noyes.) Several other cases of hemiachromatopsia without autopsies have been recorded. In one of these mental blindness, agraphia, and amnesic aphasia coexisted with right hemianopsia, the achromatopsia being of the left lateral variety. In this case all the symptoms except the right hemianopsia improved, and eventually disappeared. (Siemerling and Wilbrand.) Other cases of irregular combinations of loss of the color sense with hemianopsia, speech disturbances, and other symptoms have been recorded, all appearing to point to lesion of some part of the visual cortex as the origin of the achromatopsic disturbances.

Diagnosis, Prognosis, and Treatment of Hemianopsia.—The etiology, pathology, and diagnosis of hemianopsia have been largely considered in the preceding discussion of this visual symptom. A close study of the paragraphs relating to the site of the lesion in each of the forms of hemianopsia, and to the special associations of symptoms in the different forms, will make the focal diagnosis comparatively easy. Probably the most important single point in the differential diagnosis of the position of a focal lesion in hemianopsia is the presence or absence of Wernicke's symptom, the hemiopic pupillary inaction. If its presence is determined the hemianopsia may be certainly regarded as due to a lesion in the primary optic centres or in the tracts or nerves in front of these centres; its absence throws the lesion backward into the optic radiations or cortical visual centres. It is important to separate the hemianopsias due to focal lesions from the toxic, infectious, hysterical, or fugacious forms of this affection. When the subject of toxic amblyopias is considered, it will be seen that by far the most frequent visual defect resulting from poisoning by alcohol and other toxic agents is a central amblyopia; but in rare cases toxic or infectious agents produce hemianopsia. Hemianopsia, for example, has been observed in cases of uremia and malaria. The progress of such cases and the absence of the symptoms usually associated with hemianopsia assist in showing that a focal lesion was probably not present. Peunoff, when suffering from malaria, had an attack of complete blindness, aphasia, left hemiplegia, and hemianesthesia. Reich had right homonymous lateral hemianopsia while suffering from malarial fever. Leidy in 1889 reported to the Philadelphia Neurological Society a case in which bitemporal hemianopsia was present in a patient suffering from pronounced malaria. Harlan later made a more elaborate report on this case. The corpuscles of Laveran were present, and the patient was cured by quinine. It was at one time supposed that hysterical hemianopsia did not occur;

but a few cases have now been put on record. Landolt recorded one case, presumably of hysterical hemianopsia, but the ophthalmoscope showed abnormal findings. Glorieux reported another case of temporary right hemianopsia with right hemianesthesia in a boy sixteen years old. Rosenstein has also reported a case of right hemianopsia in a case of hysteria. (Knies.) Hemianopsia is certainly so rare in hysteria that its occurrence should lead to a careful sifting of the symptoms for the possible presence of organic disease as a factor or as a complication. Fugacious and rapidly changing forms of hemianopsia are probably due most frequently to temporary disturbances of circulation in the cell layers of the calcarine cortex, and usually are of the homonymous lateral type, but the obscuration may be of only a portion of the half field, or it may be partial at first and become complete later in the attack. Sometimes it takes the form of a hemiachromatopsia, which may be partial. Peculiar scintillations of a zigzag or bizarre character in the obscured field and phosphenes of varying intensity and of differing forms may be present. These forms of fugacious hemianopsia with phosphenes and other visual phenomena are sometimes a portion of the symptom picture in migraine. The visual symptoms may precede the migrainous attack, or may be present in its early stages. Among other evidences of disturbances of the visual cortex occasionally observed in these cases of fugacious hemianopsia are transient alexia, verbal blindness, or other disturbances of vision-speech. Oppenheim has suggested a method of examination in cases of incomplete hemianopsia—when the sensibility of the affected portion of the retina is greatly diminished but is not wholly lost. It is especially applicable in cases of bitemporal or binasal hemianopsia. Two objects of the same kind are presented to the patient simultaneously in such a way that one lies in the outer and the other in the inner half of the field of vision. When the patient is asked what he sees, he points to the object in the clear field, while the other escapes his notice. As Oppenheim suggests, the image produced by the normal retina is so vivid in comparison with the other that it blinds the mind and renders it unaware of the shadowy impression conveyed from the blunted part. The prognosis of hemianopsia will, in the first place, vary with the nature of the affection. If hysterical, if associated with migraine, or if of toxic or infectious origin, the prognosis is comparatively good. When due to a focal lesion the prognosis is that of the affection causing it, and varies according to the site of the lesion and its nature. A growth or an abscess involving some portion of the occipital lobe gives a more favorable prognosis than when the lesion causing the hemianopsia is situated at the base of the brain, as in the former case the lesion may be amenable to surgical treatment. When hemianopsia is one of the active manifestations of syphilis the prognosis is more favorable than when due to other causes. The treatment of hemi-

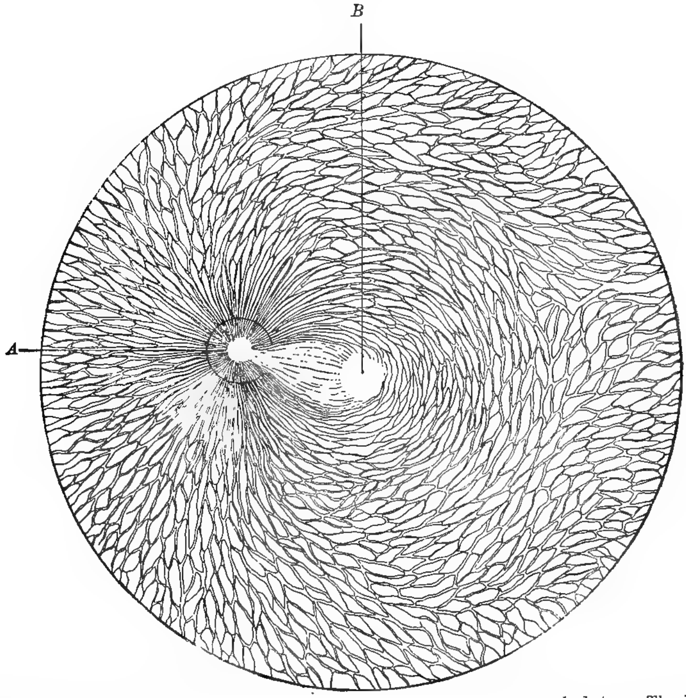
anopsia is that of its causative affections. Malarial hemianopsia, as in the case recorded by Leidy, may disappear under the administration of large doses of quinine, and hysterical hemianopsia may pass away under the influence of suggestive therapeutics and tonic medication. The surgical and medical measures intended for the relief of tumor, abscess, hemorrhage. and other focal lesions which cause hemianopsia are of course the same as those discussed when these focal affections were considered.

Diffuse Diseases of the Visual Apparatus.—*Varieties.* The preceding pages of the present section have been devoted mainly to focal lesions of the visual apparatus which most frequently claim the attention of the neurologist. The retina, optic nerves, tracts, and visual centres may also be the seats of a variety of diffuse diseases, which may be inflammatory, degenerative, or functional. The majority of these come especially under the care of the ophthalmologist, but some of them are of importance both to the general practitioner and to the neurologist. Of these affections the following will be briefly considered : *hyperemia and anemia of the retina and optic nerve, papillitis or optic neuritis, toxic amblyopias, atrophy of the optic nerve, and a few miscellaneous affections,—visual anesthesia, visual hyperesthesia, visual neurasthenia, day blindness, night blindness, and visual hallucinations.*

The Normal Eye Ground (Fundus Oculi).—Before discussing affections of the optic nerve, such as hyperemia, anemia, papillitis, toxic amblyopias, and atrophy, the diagnosis of which is largely to be made by the ophthalmoscope, the appearances of the normal fundus or eye ground should be described. The ophthalmoscope should be used in most cases of suspected disease of the cerebrospinal axis, and especially when intracranial disease is supposed to be present. By the fundus, or eye ground, as these words are commonly used, is meant the posterior interior portion of the eye as seen by the ophthalmoscope. In the first place. it is well to remember that the appearance of the fundus differs somewhat according to the complexion of the patient, and Jaeger and others have furnished colored plates showing these appearances in the brunette and other persons. In the normal fundus of the brunette the general tinge of the retina is slightly darker than in persons of lighter complexion. The main features of the landscape of the fundus are the optic disk or papilla and its immediate surroundings, and the bloodvessels, both arteries and veins. The optic disk is a yellowish white spot with a pinkish fringe, and is made up of fibres coming from all portions of the retina, which converge and pass through the lamina cribrosa to form the optic nerve. This convergence of the retinal fibres at the optic disk is shown in Fig. 370. The whitish appearance of the disk is due to the fact that the lamina cribrosa and the myelinated fibres of the optic nerve posterior to the lamina can be seen through the comparatively

transparent unmyelinated axis cylinders of the nerve proper. The actual average measurement of the optic disk is usually given as from one and four tenths to about two millimetres. It appears much larger because of the magnification produced by seeing it through the cornea and crystalline lens. It seems almost circular in outline, but in reality is slightly oval, the greater length of the ellipse being vertical. The surface of the disk, even in the normal state, is somewhat depressed, causing what is called the *physiological cup* or *excavation.*

FIG. 370.

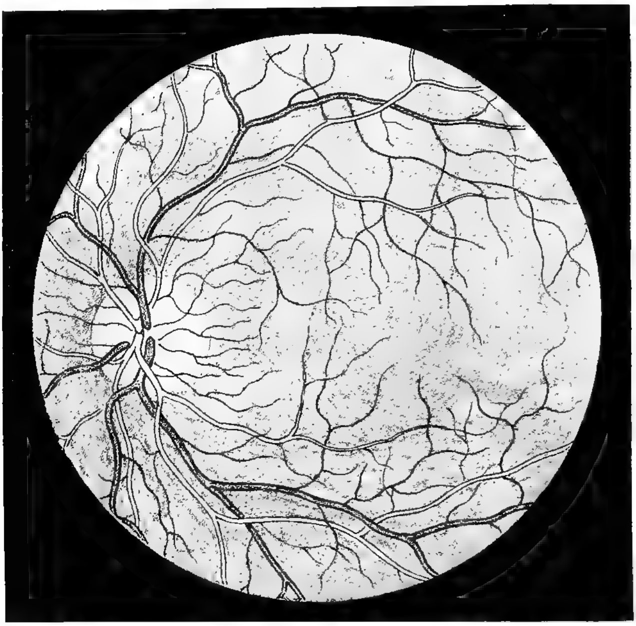


Radiation of the optic nerve fibres upon the retina: *A*, optic disk; *B*, macula lutea. The image is reversed. (Michel.)

Baker objects to the use of the name papilla because the nerve head presents a depression rather than an elevation, the apparent elevation being an optical illusion caused by the whitish nerve fibres showing through the transparent surroundings. The physiological excavation or depression is not situated exactly in the centre of the disk, but a little towards its nasal side. It varies considerably in depth, and also in shape, sometimes being almost round and at other times funnel-shaped. The depression is caused by the convergence of the fibres coming from various portions of the retina to enter the optic nerve (Fig. 370). The disk is surrounded by an inner or *scleral* ring, which is of a whitish color and represents the edge of the sclerotic coat. It is also surrounded by an outer or *choroidal* ring, which is a

dark pigmented circle. These rings are sometimes seen obscurely on the nasal side of the disk. They become strikingly changed in outline in some forms of ocular disease, as in high grades of myopia. The main artery of the optic nerve and retina is the *central artery of the retina*, which is a branch of the ophthalmic artery. It enters the retina from the optic nerve at the position of the physiological excavation. It commonly divides into a superior and an inferior branch, called the superior and inferior papillary arteries, and each of these two branches soon divides into a temporal and a nasal branch. The branches continue to divide dichotomously (Fig. 371). The methods of branching and termination of the retinal arteries are similar to

FIG. 371.



Normal eye ground of an individual with light brown hair. (Jaeger.)

those of the vessels of the cerebral cortex. Their terminals do not anastomose with one another, so that each portion of the retina is supplied by its own particular vessels. The arteries are everywhere accompanied by veins, which are a little larger than the arteries. Under normal conditions the arteries do not pulsate, but the veins may pulsate spontaneously even when the eye ground is normal. As a rule, the blood appears of a yellowish red tint in the arteries, and of a dark red or purplish color in the veins. Numerous variations and anomalies in the size of the arteries and veins of the fundus may be met with, and it is only by a considerable practical experience with the ophthalmoscope that the physician will be able always to see when the vascular appearances are absolutely normal. Those por-

tions of the eye ground which are removed from the optic disk usually present a yellowish or brownish red color. The macula lutea, the seat of most acute vision, is an oval spot, somewhat darker than other portions of the retina, owing to an excessive amount of pigment granules diffused throughout its tissues. The *fovea centralis*, the central portion of the macula, is situated almost exactly in the axis of the ocular globe, at an average distance of about 3.915 millimetres outward from the centre of the disk. The normal appearances of the fundus are different at different ages, these differences being especially marked between the period of childhood or youth and that of old age. For a fuller description of the appearances of the normal fundus, works on ophthalmology should be consulted.

Hyperemia of the Retina and Optic Nerve.—*Etiological Varieties.* Hyperemia of the optic nerve and retina is comparatively rare as a clearly recognizable and isolated affection, although its presence is frequently diagnosticated without due consideration. A true hyperemia is present in the early stages of papillitis or optic neuritis, but the progress of such a case soon shows its true nature, the optic disk in a short time visibly swelling, while changes take place which make its structure less transparent and obscure its borders. The hyperemia which results from intraocular disease such as iritis, and that which is due to such causes as exposure to heat or glare, fall exclusively under the care of the ophthalmologist, and will not be here discussed. The forms of hyperemia of the retina and optic nerve which most concern us are associated with various diseases of the cerebrospinal axis, and especially with irritative diseases, like intracranial tumor, abscess, and meningitis. These affections are, as a rule, associated with a true papillitis, but in some instances give rise only to a retinal and neural hyperemia. Occasionally in cases of brain tumor decided venous engorgement without swelling of the disk or blurring of its edges is present. In epilepsy congestion of the retina and optic disk has been noted, and a low grade of congestion is sometimes chronic. An acute hyperemia may be the immediate result of a convulsive attack, or it may be produced by disease of the heart, or by any other cause which leads to engorgement of the vessels of the head, as, for instance, obstruction of the vessels of the neck. Violent coughing or sneezing may cause transient but comparatively severe forms of hyperemia. The condition of the vessels of the optic nerve and retina in various forms of insanity has been carefully studied by different observers. Wiglesworth and Bickerton hold that, in the first place, it is necessary to draw a clear distinction between general paralysis of the insane and other forms of insanity, in arriving at conclusions as to the ophthalmoscopic appearances in the insane. They conclude that in insanity proper, including general paralysis of the insane, no connection can be traced between the condition of the fundus and the patient's

mental state. In a minority of cases of general paralysis of the insane, clear and precise ophthalmoscopic lesions were found, these cases falling into two classes: cases which tend to develop isolated neuritis, and cases tending to pass into optic atrophy. In the former of these classes the affection declares itself as a hyperemia of the disk. The hyperemia and neuritis tend, if the patient lives long enough, to be replaced by atrophy. Allbutt observed hyperemia in mania, dementia, and general paralysis of the insane. Various observers have remarked a similar condition in the early stages of general paralysis of the insane, and it has even been recorded as present in melancholia. Lautenbach found retinal hyperemia in forty per cent. of the acute cases examined by him, his entire investigation including over seven hundred cases of insanity of different forms. Some infectious diseases and certain toxic agents, such as alcohol, tobacco, lead, and arsenic, give rise sometimes both to hyperemia and to inflammation of the optic nerve, a subject which will be more fully considered under toxic amblyopias. A true hyperemia which may pass into a low grade of inflammation is an accompaniment of some disorders of refraction which cause almost constant eyestrain. As all efforts at convergence and accommodation are followed by increased flow of blood to the eye, abnormal and prolonged efforts must tend to produce congestion and even inflammatory states.

Symptomatology, Diagnosis, Prognosis, and Treatment. Only a thoroughly competent ophthalmologist can determine the presence of a true congestion of the fundus unassociated with inflammation. Even the existence of a degree of redness in excess of that which is normally found in the nerve is not a proof of the presence of true hyperemia; but when, instead of the yellowish white appearance of the nerve, with its pinkish border, the disk assumes a dull red or brick-dust color, and when the edge of the disk is so obscured that it can only with difficulty be distinguished from the general eye ground, the condition may be regarded as one of true hyperemia. (Gowers.) In some cases the hyperemia is monocular, when a comparison of the two eye grounds will make the diagnosis much more easy. If the condition is one of hyperemia and not a true inflammation, the papilla will not project into the eye, and hemorrhages will not be present. A skilful observer can make out the borders of the disk, although these may be somewhat obscured. Unusual tortuosity of the veins is present in hyperemia, as in neuritis; but too much stress must not be laid upon mere tortuosity of the veins. The diagnosis of hyperemia of the retina and optic nerve is therefore to be reached by making a critical distinction between the appearances presented by the normal fundus, those of inflammation in different stages, and those which have been just described as characteristic of genuine hyperemia. The prognosis of hyperemia will depend largely upon the disease with which it is associated; when present as an inde-

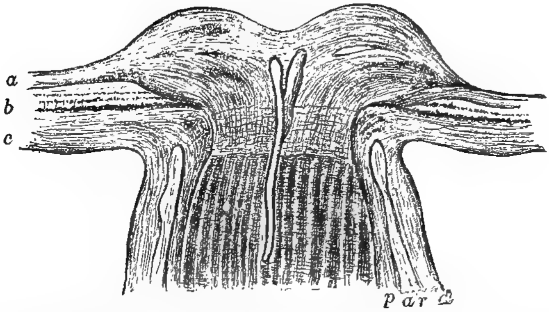
pendent affection the prognosis is usually comparatively good, the hyperemia disappearing with the removal of the causes which have led to its presence or continuance. The treatment will usually be that for abscess, brain tumor, meningitis, encephalitis, general paralysis of the insane, and the other affections with which it is ordinarily associated. In simple hyperemia of unknown origin the eye should be kept at rest as much as possible, and remedies like local bloodletting, bromides, and ergot may be used. Tinted glasses are sometimes required to protect the eyes from unusual glare.

Anemia of the Retina and Optic Disk.—Anemia of the retina and optic disk accompanies general anemia, and also at times certain forms of insanity and certain focal diseases of the brain, such as embolism. Simple anemia was one of the four abnormal conditions of the optic disk found by Wiglesworth and Bickerton in their examinations of the eye grounds of the insane. Anemia of the retina has been noted in melancholia and chronic mania. It is necessary to distinguish simple anemia of the optic disk from the pallor of the early stages of atrophy, but when the atrophy has made some advance this difficulty disappears. Sudden retinal anemia is sometimes caused by occlusion of the central artery of the retina, or the same result may be brought about by compression of this vessel. Anemia from spasm of the retinal vessels is also said to occur in cases of acute poisoning from quinine, and in these cases more or less contraction of the visual fields may remain. The ophthalmoscope shows pallor of the disk and attenuation of the vessels of the fundus.

Optic Neuritis or Papillitis.—*Symptomatology.* Optic neuritis or papillitis is a subject of great importance both to the neurologist and to the ophthalmologist, but has already been largely considered in this work when discussing meningitis, tumor, and abscess. It has been especially discussed under encephalic tumors. (See pages 510, 511, also 521, 522.) The presence of papillitis or optic neuritis is to be determined by the use of the ophthalmoscope and by a comparison of the known appearances of the nerve when normal and when hyperemic, anemic, or atrophic. The appearances presented by the nerve in a well marked case of papillitis are shown on page 511, Fig. 294. As the papillitis develops, the nerve head becomes red and swollen and soon loses its normal contour. Its edges become indistinct and are gradually entirely obscured. The tissues are infiltrated and present hemorrhagic spots; the veins are increased in size, while the arteries become shrunken. The nerve head is sometimes so enormously swollen as visibly to project into the eye. It may reach the size of three millimetres, although it is commonly much less than this. Vision is affected very irregularly. Sometimes it is rapidly lost, more frequently it slowly diminishes. Occasionally it is preserved in a remarkable manner even with extreme swelling of the disk, and in rare instances it may for a time be

more acute than normal. The field for form is usually concentrically contracted, color changes commonly taking place early. As a rule, the perception of red and green is lost first. In what is usually termed choked disk the inflammation is more intense, the papilla is more swollen, and hemorrhages are more frequent. Edema both of the disk itself and of the surrounding retina is present, while the

FIG. 372.



Longitudinal section through the head of the optic nerve, showing choked disk: the optic disk is swollen and the vessels are engorged: *a*, the retina; *b*, the choroid; *c*, the sclera; *d*, the dural sheath; *p*, the pial sheath; *ar*, the arachnoid space. (Wilder.)

vessels are greatly engorged. In Fig. 372 is shown a longitudinal section through the head of an optic nerve presenting the condition known as choked disk.

Theories as to the Nature of Optic Neuritis. Reference has been made to the Leber-Deutschmann theory of the pathology or method of causation of optic neuritis, which the writer regards as most probably correct. Several other theories concerning the mechanism of optic neuritis, however, have been advanced, and for a full discussion of these, works on ophthalmology should be consulted. Recently Parinaud has revived the old theory that papillitis is due to an edema of the trunk of the optic nerve, similar to the edema which occurs in brain tissue, the later inflammation of the retina, so common in optic neuritis, being caused by the presence in the nerve of extraneous material. He believes that excessive intracranial tension is incapable of producing edema of the papilla, and that edema of the nerve does not require any considerable excess of this tension nor mechanical damming up of fluids in the nerve. He suggests calling the neuritis of intracranial origin *edematous neuritis.*

Etiology. Papillitis is most frequently associated with such focal intracranial diseases as tumor, meningitis, or abscess. Its special features, and the frequency of its occurrence in these affections, have already been sufficiently considered. It is occasionally present in myelitis. It occurs also in poisoning from lead, arsenic, alcohol, and other drugs, and in the course of some of the infectious diseases, as scarlet fever, measles, diphtheria, typhoid fever, and influenza. It

is sometimes observed in the course of disorders which greatly impair nutrition, as in pernicious anemia, leucocythemia, diabetes, and Bright's disease. In the last affection the ophthalmoscopic appearances are often of a special character (see page 522). It is somewhat frequently present in syphilitic disease, even when intracranial gummata cannot be diagnosticated. Syphilis may undoubtedly attack the optic nerve directly, leading either to neuritis or to noninflammatory degenerations. Acute monocular optic neuritis has been observed as the result of gonorrheal infection. (Highet and Reiss.) Acute rheumatic optic neuritis is occasionally seen, and is important from prognostic and therapeutic points of view. It is often monocular, follows exposure, and is characterized by sudden onset. In these cases papillitis is sometimes typical, and, unless the neuritis be efficiently attacked, white atrophy may result. (Zimmerman.) In rare instances optic neuritis seems to be an inherited or family form of disease; and in reported cases of this kind it has usually attacked male members of the family at about the age of twenty years. Neuritis may occur as a purely local disease from inflammations, tumors, or other lesions in the orbit. Most of the causes above mentioned may give rise to an acute, subacute, or chronic optic neuritis. Acute retrobulbar neuritis most frequently results from lesions in the orbit, or from infectious and constitutional or diathetic processes. Toxic agents, like alcohol, occasionally give rise to severe forms of acute retrobulbar neuritis, but more frequently they produce the chronic forms of this affection,—the toxic amblyopias. W. H. Wilder reports a case in which he believes excessive weeping produced disturbances of circulation and excited the neuritis. When an optic neuritis arises apparently without cause it is probable that some unknown infection or toxemia is the source of the disease. Overwork, physical or mental, is an assigned cause.

Diagnosis, Prognosis, and Treatment. The diagnosis is chiefly to be made with the aid of the ophthalmoscope, although photophobia and disturbances of vision may cause the neuritis to be suspected. Low grades of inflammation which scarcely deserve the name of neuritis are observed in a comparatively large number of cases of abnormal refraction. According to Norris, if the swelling of the papilla is two diopters or over, intracranial or intraorbital disease is the probable cause. This is an important diagnostic point in differentiating papillitis due to intracranial or constitutional causes from the inflammation which accompanies disorders of refraction. In the latter a swelling of from one to one and a half diopters can sometimes be determined. The prognosis in the vast majority of cases of optic neuritis associated with intracranial disease is unfavorable, although the inflammation may improve after trephinings and even when growths are not removed. In those cases in which the papillitis is due to infectious or toxic agents the prognosis is relatively more favor-

able, depending somewhat upon the activity of the treatment, but even in these cases the inflammation frequently goes on to atrophy. In a considerable percentage of the cases of acute retrobulbar neuritis a partial or complete cure is effected. In the neuritis associated with disorders of refraction the prognosis is relatively good. The treatment of optic neuritis is chiefly that of the disease or condition with which the affection is associated, as tumor, meningitis, abscess, rheumatism, diabetes, syphilis, and the other affections which have been mentioned in the discussion of etiology. General rest, absolute rest of the eyes, and protection from glare are of great importance. The local abstraction of blood, the use of derivatives to the head and the temples, and other local measures, may be required. Repeated mercurialization and the iodides in syphilitic and sometimes in non-syphilitic cases; the salicylates when a rheumatic cause is suspected; and iron, arsenic, peptomangan, and other blood tonics in anemic and diabetic cases, are among the remedies naturally suggested.

Toxic Amblyopias.—*Definition and Varieties.* Toxic amblyopias are affections in which vision is obscured or lost as the result of the action of toxic agents taken into the system.* The lesions produced by these agents are sometimes distinctly limited to portions of the optic nerves, but in other cases they may involve the retina and other more central portions of the visual apparatus. Toxic agents may affect any portion of the optic-ocular mechanism; thus, they may produce either mydriasis or myosis, through their influence upon the pupillary mechanism. Our only concern in the present connection is with those poisons which directly affect the optic nerve and retina, and especially with those which produce certain peculiar disturbances of central vision. The toxic amblyopias may be either acute or chronic; but the latter have received the most attention.

Toxic Agents which most frequently cause Amblyopia. The two drugs which hold the first place in the production of toxic amblyopias are *alcohol* and *tobacco*. Amblyopias are also of comparatively frequent occurrence as the result of the use of *quinine* and *lead.* Cinchonidia salicylate, sodium salicylate, and salicylic preparations in general, when given in large doses or when taken by persons with idiosyncrasies, may produce toxic effects upon the visual apparatus similar to those caused by quinine. When the optic nerve is toxically affected by lead, the entire bulk of the nerve is likely to be involved. Alcohol has been known as a cause of amblyopia since the time of Boerhaave (1751), but special attention has been directed

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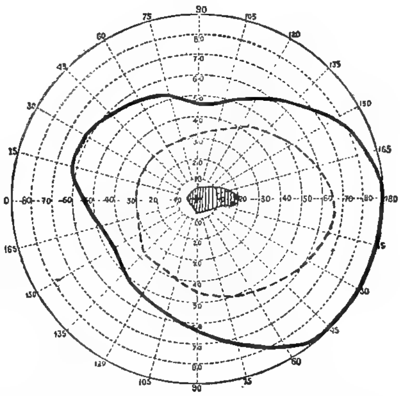
* The most important recent work on *Toxic Amblyopias* is by Professor George E. de Schweinitz, M.D., of Philadelphia. This work, which has been used in the preparation of the present section, discusses the subject in all its aspects, reviewing the literature and giving the experience of the author. The fields shown in Figs. 373, 374, and 375 were kindly furnished by Professor de Schweinitz.

to the subject only in recent years. Alcohol and tobacco sometimes act conjointly in causing amblyopia, and some have even doubted the exclusive action of alcohol in its production. It is clear, however, that alcohol alone is responsible for a considerable percentage of amblyopic cases. Doubtless tobacco on the one hand and alcohol on the other are more efficient when both narcotics are used by the patient. By some authorities the presence of fusel oil is regarded as essential in the production of alcoholic amblyopia; but, while impurities may add to the deadly effects of the drug upon the optic nerve, the alcohol alone is sufficient. Alcoholic or tobacco amblyopia may be either acute or chronic, but it is much more frequently the latter. It has been made clear by numerous exact observations that a form of central amblyopia is due to the use of tobacco alone, and the tobacco may be introduced into the system in a variety of ways; most frequently the amblyopia is caused by smoking and chewing, but it may be brought about in other ways, as by the inhalation of the vapors of tobacco or by contact with decoctions of this drug.

Miscellaneous Drugs causing Amblyopia and other Visual Disturbances. It has been asserted that both optic neuritis and atrophy have resulted from mercurial poisoning, but the recorded cases are not of a decisive character. In a table of one hundred and thirty-eight cases by Uhthoff, two are attributed to the poisonous action of carbon bisulphide. Cases of amblyopia from the action of silver nitrate have been recorded, and in these cases silver has been found in the sclerotic sheath of the optic nerve. In phosphorus poisoning fatty degeneration of the optic elements sometimes takes place. Iodoform, arsenic, and nitrobenzin may produce optic as well as other forms of peripheral neuritis. Carbolic acid, naphtalin, and various coal tar products also cause visual disturbances, the pathological nature of which has not been demonstrated. Central amblyopia has apparently been produced by chronic stramonium poisoning. The anesthetic, hypnotic, sedative, and depressant drugs may all produce affections of vision, but evidence as to the exact manner in which they exert their influence is wanting. Cases have been reported in which potassium bromide in

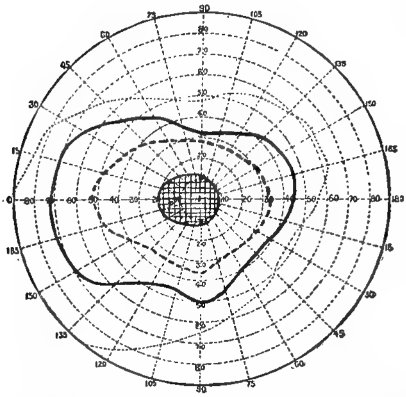
FIG. 373.



Average general size of scotoma (right eye) in toxic amblyopia; normal peripheral fields for form and red shown respectively by solid and dotted lines. Scotoma mapped with two millimetres square of red, the eye being placed thirty centimetres from fixation.

large doses has produced amblyopia with whiteness of the optic nerve, which disappeared when the drug was withdrawn and reappeared when it was again administered. Digitalis, atropine, hyoscine, homatropine, daturine, duboisine, cocaine, gelsemium, eserine, and conium exert their influence chiefly upon the oculopupillary apparatus, the optic nerve not being directly affected.

FIG. 374.

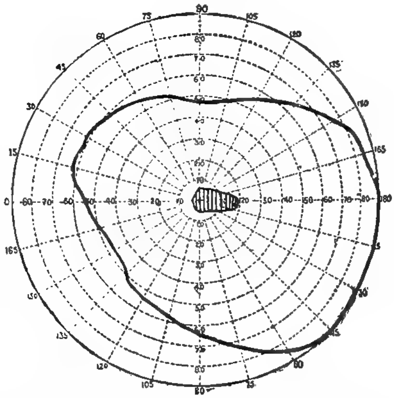


Large scotoma (left eye) in a case of non-toxic retrobulbar neuritis; some contraction of form and blue fields as shown respectively by the solid and inner dotted lines; green was not recognized, red doubtfully.

Amblyopias from Diathetic and Infectious Diseases. In much the same manner as toxic agents, diathetic and infectious agents like syphilis, diabetes, gout, rheumatism, influenza, and malaria may cause amblyopia. Diabetes causes not only retinitis and cataract, but also in some instances degeneration of the optic nerve with central scotomas. Optic neuritis and neuroretinitis resulting from malarial poisoning have been observed especially in countries where pernicious malaria is common. In some of these cases the patients become totally blind, and the optic disks are obscured by effusions which may extend into the retina. The affections are improved, or even cured, by treatment with quinine, arsenic, and strychnine.

Symptomatology. In alcoholic amblyopia, in the first place, such general symptoms of chronic alcoholism as tremor, insomnia, irritability, restlessness, gastro-intestinal disturbances, peripheral neuritis, chronic or subacute myelitis, mental deterioration, or insanity, may be present. Optic neuritis may develop either independently or in association with a slight meningitis. The ocular symptoms, as summarized by Uhthoff and de Schweinitz, are pathological whiteness of the temporal half of the optic papilla, occasional haziness of the nerve head or hyperemia of its surface, and rarely retinal hemorrhages.

FIG. 375.



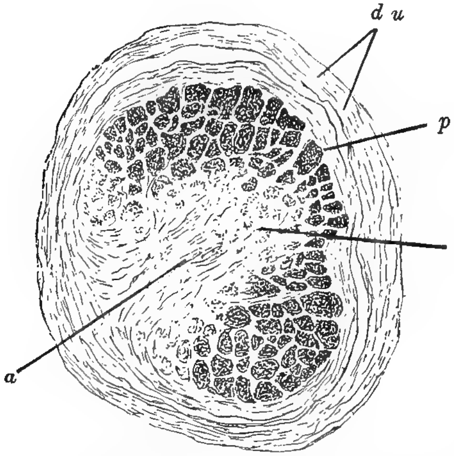
Scotoma (right eye) in a case of tobacco-alcohol amblyopia; patient forty-nine years old, smoked since seventeen, averaging twelve cigars a day; also drank whiskey freely; complete cure after stopping tobacco.

The whiteness of the temporal half of the papilla, or sometimes only of a quadrant in the lower and outer portion, is the most frequent sign. In the early stages the field of vision shows a relative scotoma for red and green, and sometimes complete or partial peripheral defects for the same colors. Small central scotomas for blue may be present in rare cases, and various unusual conditions of the central color fields are exceptional. Visual acuity may vary from one third of normal vision to one fortieth, and pupillary phenomena are unimportant. (De Schweinitz.) The presence of central scotomas is the most important feature in alcoholic as in tobacco amblyopia. Efforts have been made, but without decided success, to establish a differential diagnosis between alcoholic and tobacco amblyopia and the mixed types of amblyopia, by the forms of the scotomas. Various general symptoms, physical and mental, may be present early in cases of tobacco amblyopia, or indeed may manifest themselves before the amblyopia is observed. In other cases, perhaps the majority, the amblyopic symptoms are the first to attract attention. The general symptoms are such as impairment of appetite, insomnia, deficiency in memory and mental concentration, restlessness, irritability of the heart, and disturbances of the digestive apparatus. So far as the visual symptoms are concerned, as a rule the patient's first complaint is of a dimness of vision, which he soon learns cannot be improved by glasses. The centre of the field is most obscured, and the patients see best in a somewhat dull light. The acuity of vision is usually very markedly diminished when the patient first comes under observation. The pupils show no positive changes, except perhaps a tendency to dilatation after the affection has become chronic. The external ocular muscles are not affected. The ophthalmoscopic appearances of the disk are not absolutely diagnostic.

Pathology and Morbid Anatomy. The origin of toxic amblyopia is an interstitial inflammation, commonly a *chronic retrobulbar neuritis*, which in time is followed by atrophy of the true nerve elements. In an alcoholic case with all the classical symptoms of toxic amblyopia Samelsohn found atrophy of the papillomacular bundle, and other similar cases have been put on record. Uhthoff has recorded similar pathological findings in six cases of undoubted alcoholic amblyopia. The inflammatory changes are found especially in the retro-orbital portion of the nerve, and as the nerve and tract are traced backward simple degeneration of the macular bundle is the important pathological condition. In tobacco amblyopia the connective tissue is thickened at an earlier period than in alcoholic cases. The nerve elements almost entirely disappear in the former, while in alcoholic atrophy some remnants of the nerve fibres can usually be discovered. The central degeneration occurring in cases of toxic amblyopia is shown in Fig. 376.

Diagnosis, Prognosis, and Treatment. The diagnosis of toxic amblyopia is to be made by a careful study of the history of the patient with regard to the abuse of alcohol and tobacco; by an investigation of the fields of vision for central scotomas; by the ophthalmoscopic appearances, which are sometimes significant; and by a search for the other evidences of toxic disease. The prognosis is variable. In a large number of cases it is relatively good if the patients can be induced to stop the use of the drugs which have caused the affection. Under the measures to be presently described, patients sometimes make rapid and at other times slow but steady improvement. In some cases, however, the nerve inflammation goes on to incurable atrophy. The patient's general condition and powers of resistance play some part in the problem of prognosis. In the treatment of toxic amblyopia the matter of first importance is, of course, the removal of the causative agent. The alcohol or tobacco, or both, must be discontinued; and this is by no means an easy matter to accomplish with patients who have for years been addicted to these narcotics. Some results seem to have been achieved by the use of eliminatives like potassium iodide, ammonium chloride, and free diaphoresis. The most important drug in the treatment of toxic amblyopia is strychnine. It can be used either by the mouth or hypodermatically, or in both ways at the same time. As a rule, it is best to use the drug in very decided doses, although the treatment may be begun with small doses, these being gradually or rapidly increased to a maximum. Thus, strychnine nitrate can be administered hypodermatically in doses of one forty-eighth of a grain and increased from day to day or every other day until the patient is taking as much as one tenth of a grain daily, or even more. At the same time moderate doses of either nux vomica or strychnine sulphate can be given three or four times daily by the mouth.

FIG. 376.



Cross-section of the optic nerve about one millimetre behind the eyeball, showing marked atrophy chiefly in its central fasciculi. The healthy nerve fibres are stained black, while the atrophied portions of the nerve, like the connective tissue of the sheath, are colored a light yellowish brown: *a*, the most atrophied portion of the nerve; *b*, bundles of fibres that have undergone partial atrophy; *du*, dural sheath; *p*, pial sheath. (Wilder.)

Atrophy of the Optic Nerve.—*Definition and Varieties.* The optic nerve, as the result of various pathological actions, undergoes

certain retrogressive and degenerative changes which are classed under the general head of *optic atrophy*. The nerve elements waste and disappear, and secondary pathological conditions result in all portions of the visual apparatus. Two most important varieties of optic atrophy are recognized by all authorities, namely, *primary optic atrophy* (*primary degeneration*) and *postneuritic* or *consecutive optic atrophy*. The latter form is sometimes called *secondary optic atrophy*, but this term is also applied to a class of cases in which optic atrophy results from pressure but is not consecutive to an inflammation of the nerve. Probably the term secondary noninflammatory atrophy is the best to apply to this third class of cases, as to the nature of which opinions differ. When the term secondary atrophy is applied to postneuritic cases, it should be so qualified as to indicate its origin.

Primary Optic Atrophy.—*Associations, Etiology, and Pathology.* Primary optic atrophy would perhaps be best discussed at length under primary degenerations of the nervous system, such as tabes, disseminated sclerosis, lateral sclerosis, dementia paralytica, and other similar affections, in association with which by far the largest number of cases are observed. It is necessary, however, briefly to discuss the subject here in order to complete our survey of the affections of the visual apparatus. Too often this form of atrophy is considered as consecutive to the cerebrospinal lesions of these more general diseases; but it should be regarded rather as one of the phases of an inherent tendency to primary degeneration. Numerous recorded cases show that the optic nerve may suffer at greatly differing periods in the course of such a disease as tabes. The atrophy may be an early, a late, or an intermediate symptom in tabes, or it may never be present, even in a case of the most serious type. On the other hand, optic atrophy may for a long time be the only recognized symptom of such disease; and in exceedingly rare instances the disease may be limited throughout its manifestations to optic atrophy. With Dr. S. D. Risley, of Philadelphia, and other ophthalmologists, I have studied a number of cases of tabes in which primary optic atrophy at first, or even for a considerable period, was the only symptom that attracted the attention of either patient or physician. The atrophy may precede the other symptoms for several years. It has been observed seven, ten, fifteen, and twenty years prior to any other tabetic symptoms. Primary optic atrophy is in rare cases congenital and of hereditary origin. Among other rather uncertain causes some authors mention the influence of cold, menstrual disorders, and venereal excesses. Profound anemia appears sometimes to give rise to optic atrophy, as to degeneration of other portions of the nervous system. Like the diseases with which it is so frequently associated, it occurs much more frequently in men than in women. It may develop at any age, but it is oftenest observed at and after middle life. It is an interesting clinical fact, emphasized by Charcot, that when

it occurs with disseminated sclerosis it does not usually progress to total blindness. The examination of the optic nerve in a case of primary atrophy shows it to be shrunken and sometimes more or less gelatinous. In the progress of the affection the nerve fibres lose their sheaths and later become granular and fatty.

Symptomatology. The vision of the patient gradually becomes very indistinct,—in some cases so imperceptibly that unless his occupation is of such a kind as to make him critical of his exact visual powers its progress may not be noted. As the disease progresses, objects appear misty or half veiled. Gradually also the field of vision is contracted, beginning most frequently on the temporal side. Great irregularities in the loss of vision as well as in the alterations in the fields for form and color may be shown. Commonly the peripheral field is lost before the central is attacked. Central or irregularly situated scotomas may be present, or the contractions of the fields may be irregular and much like those seen in hysteria. The fields for form and color are usually affected together, but very considerable atrophy, as determined by the ophthalmoscope, may be present without the changes in color perception and in the fields which would be anticipated. The field for colors may be greatly changed before that for form is affected. Red and green are among the first colors to be lost, while blue and yellow may still be discriminated. It is a point important to remember that nearly total gray atrophy of the optic disk may be shown by the ophthalmoscope with scarcely any disturbances of vision. The distinct ophthalmoscopic appearances can be recognized at an early stage by an expert ophthalmoscopist. They are more or less significant at all stages. When the disease has made a little headway the outline of the disk is usually sharp. The laminæ are commonly visible and mottled. The color of the disk is a reddish gray, or gray, and the discoloration is usually first visible on its outer half. In time it extends over the entire nerve head, which acquires an excavated appearance. The details on the surface of the papilla, especially the tendinous network of the lamina cribrosa, remain clearly visible as in glaucomatous atrophy, and in contrast with postneuritic white atrophy, in which the meshes of the lamina cribrosa are invisible or very indistinct. (Knies.) Inflammatory phenomena are absent. In the early stages the vessels do not show contraction. Late in the affection atrophy occurs in the finer connective tissue bands within the larger meshes. The ganglion cells and nerve fibres of the retina eventually atrophy. Atrophy of the eyeball sometimes occurs, giving a form of microphthalmus. Among special symptoms of advancing atrophy which have been noted are frontal or temporal headache, pain in the eyes, phosphenes, and a species of photophobia, which shows itself by a feeling of discomfort or irritation produced by glare.

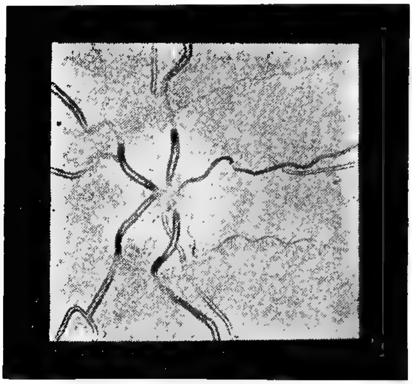
Diagnosis, Prognosis, and Treatment. The diagnosis is to be made

by careful consideration of the symptomatology and of the ophthalmoscopic appearances as just given. Whenever progressive failure of sight is observed without evidence of focal disease of the brain or of toxemia, the probability of the existence of primary optic atrophy should receive consideration. In such cases also it is always well to investigate for the presence of other symptoms of neuraxial degeneration, such as lancinating pains, altered knee jerks, disturbances of the bladder, and ataxia. Phenomena of this kind may be present and may not have attracted special attention. Often the ataxic pains are regarded as rheumatic, while the absent knee jerk has been overlooked. The course of primary optic atrophy is almost invariably towards complete blindness. As a rule, it occurs in from one to two or three years after the atrophy is first noted; but only a few months may be required. On the other hand, many years may elapse. Gowers reports one case in which blindness was not complete until seventeen years after the first appearance of atrophy. No treatment is of any permanent avail against a disease which is necessarily fatal to sight, but the course of the affection may sometimes be arrested and vision be retained for a longer period under the use of a few remedies, notable among which are preparations of strychnine, which should be used in increasing doses up to the maximum amount which the patient can stand. Hypodermatic injections seem to give the most decided results. The silver and gold salts are of value in optic atrophy, just as they sometimes are in improving the spinal symptoms of tabes. Almost any form of tonic or nutrient may be of service by improving the patient's general condition. Benefit has been claimed from the use of suspension, but without any just foundation. Some transient improvement has been obtained by the use of galvanism. At the most, all that can be achieved by any treatment is the improvement of the patient's general condition and the temporary attainment of a slightly higher visual acuity. Most of the benefit in such cases is seeming rather than real. Culbertson has reported two cases of optic atrophy in which, after strychnine had produced only partial benefit, nitroglycerin in doses of one one-hundredth to one one-hundred and fiftieth of a grain twice daily effected marked improvement in vision.

Postneuritic Atrophy.—Postneuritic or consecutive atrophy of the optic nerve follows inflammation of the nerve. In a large majority of cases of papillitis the condition passes into a partial or complete optic atrophy. In some cases the impairment of sight is not marked until such atrophy has made considerable advance. The symptoms of postneuritic atrophy, unlike those of primary degenerative atrophy, are associated with the phenomena of intraocular inflammation, and frequently with those of active encephalic lesions. Special irregularity in the method of impairment of sight may be observed. Even when the acuity of vision is not much impaired,

examinations with the perimeter may show alterations and irregularities in the fields for form and color. As the case progresses, the loss of vision and the inability to appreciate colors become more and more marked and irregular. Examination with the ophthalmoscope gives different appearances according to the stages of both the inflammation and the atrophy (Fig. 377), the fundus at certain periods necessarily presenting an admixture of both inflammation and atrophy, the chief signs of the former being some remaining swelling of the nerve head and obscuration of the vessels. Even when the case first comes for examination, after complete blindness and extreme atrophy have occurred, the fact that the affection is consecutive or postneuritic can sometimes be recognized with the ophthalmoscope. Opaque, jellylike whiteness of the disk, irregularity or indistinctness of the margin, covering up of the region of the vessels, the presence of white lines along the sides of the vessels or of white objects on the surrounding retina, and the fact that the size of the vessels is markedly different from the normal, are in favor of the postinflammatory character of the atrophy. (Bramwell.) Occasionally, after an attack of papillitis and after partial atrophy has taken place, the inflammation having subsided, a new attack of neuritis is imposed upon the atrophic nerve, giving ophthalmoscopic appearances of a peculiar character. Postneuritic atrophy is most frequently associated with encephalic tumor, meningitis, or abscess; but in a few cases a primary inflammation of the retina and nerve may be followed by complete atrophy and blindness. These cases are usually due to infectious or toxic causes. The changes in the nerve vary according to the stage of the process. Late, in extreme cases, the true nerve elements may have disappeared, leaving fibrous tissue. The diagnosis has to be made by the ophthalmoscopic appearances and the facts which can be determined with regard to the concomitant affections; but occasionally a postneuritic atrophy is arrested before complete destruction of the nerve has taken place. When papillitis was discussed it was stated that it sometimes subsided spontaneously or under active medicinal treatment; also that it subsided or improved after removal of growths by surgical operation, or even in some cases after simple trephining without the removal of tumors. Doubtless in some of these cases atrophy has begun and its progress is to a certain extent arrested, but in other

Fig. 377.



Partial postneuritic atrophy. (Gowers.)

cases, even though the growth is removed or the inflammation subsides after an operation, the atrophy when once initiated may go on to complete extinction of the nerve. No treatment is of service for the restoration of sight in cases of complete or nearly complete consecutive atrophy. The only hope is that it may be arrested when in the partial stage and when associated with disappearing inflammation.

Noninflammatory Secondary Atrophy.—In speaking of the varieties of optic atrophy it was stated that the term noninflammatory secondary atrophy is the one most applicable to a class of cases distinct in causation from both the primary degeneration and the postneuritic or consecutive atrophy. These cases are due to pressure on the optic tracts, as by a tumor, aneurism, abscess, exostosis, or other pathological process capable of exerting pressure. This form of atrophy may also result from injury to the nerves or tracts. Secondary atrophy or involution is the almost necessary result of such pressure or injury. Vision may be lost rapidly or slowly. Not infrequently both postneuritic and noninflammatory secondary atrophy coexist in the same case, the lesion which causes pressure at the same time giving rise to inflammation. Both meningitis and neuritis may radiate from the site of a tumor which also more or less completely obliterates the optic tract in the process of its growth. The ophthalmoscopic appearances in cases of pure noninflammatory secondary atrophy differ from those which are typical both of primary optic degeneration and postneuritic atrophy. They differ from the latter in the absence of signs of true inflammation in the early stages of the atrophy. Late in the progress of the cases the appearances are very similar to those which are found in primary optic atrophy, but even here a distinction can be made on close investigation. The disk, while resembling that of primary degeneration, in this form of atrophy is more frequently distinctly white; both the veins and the arteries are contracted, the veins usually proportionately more so than the arteries. (De Schweinitz.) When postneuritic and noninflammatory secondary atrophy coexist, the opththalmoscopic appearances in the early stages are those both of inflammation and of atrophy, and it is difficult to draw from the ophthalmoscopic examination any conclusions as to the nature of the process causing the atrophy. Sufficient has been said about the etiology and nature of this form of atrophy; its symptomatology, diagnosis, prognosis, and treatment are practically the same as those of the other forms of atrophy. The removal of an intracranial tumor or the evacuation of an abscess might check the progress of a consecutive atrophy, leaving the patient with some vision, which could be improved by the administration of strychnine. When compression of the nerve and descending atrophy are due to exostosis which results from a syphilitic ostitis or periostitis of the optic canal, active antisyphilitic medication might arrest the atrophy by removing its source.

Miscellaneous Visual Affections.—*Visual Anesthesia and Hyperesthesia.* Among retinal disturbances which occasionally come under the notice of the neurologist are visual anesthesia and hyperesthesia, including night blindness (*nyctalopia* or *hemeralopia*) and visual asthenopia. The full discussion of this subject belongs properly to works on ophthalmology. Night blindness may be due to either peripheral or central disease, and it is often associated with inflammatory or nutritional changes in the retina or the choroid. Patients with retinal hyperesthesia can use their eyes best in moderate light. After a night's rest or after they are refreshed and invigorated by a meal the retina responds much less morbidly and can be used with less fatigue and discomfort. Photophobia is sometimes extreme in these cases. Anesthesia of the retina is not of infrequent occurrence as an hysterical phenomenon, especially in women and children about the period of puberty or between puberty and adolescence. In retinal anesthesia there is diminished and often qualitatively changed action of light upon the percipient retinal elements, or diminished conducting power of the optic nerve, or both; in night blindness and torpor of the retina there is, in the main, only the former condition. (Knies.)

Asthenopia not due to Refractive Errors. Gradle, in a study of five hundred patients with asthenopic complaints, found hypermetropia, astigmatism, or presbyopia in from seventy-five to eighty per cent. of the cases. In from twenty to twenty-five per cent. the asthenopia could not be accounted for in this way. Either the optic conditions were perfect, or their correction gave no relief. Under the name of *normal asthenopia* he defines that amount of fatigue and discomfort which is inevitably produced by refractive error when this is of sufficient degree, while he designates as *exaggerated asthenopia* the excessive eye annoyance which fatigue and eyestrain lead to only in certain persons. The underlying factor in the production of the latter is inherited or acquired weakness of the nervous system; but infectious disease, anemia, indigestion, faulty habits, improper hygiene, and especially insufficient muscular work, may be factors in its production. The interesting suggestion is made by Gradle that a few cases are due to anomaly of the retinal pigment epithelium. The fundus shows in these cases what is described as graining or salt and pepper appearance. He believes that it is not identical with Gould's ametropic choroiditis. With a few exceptions, the children in whom he found this appearance of the fundus were asthenopic. The only therapeutic measures which gave any relief were absolute rest of the eyes and dark glasses. This writer, contrary to a large number, if not a majority, of ophthalmologists, believes that the number of cases in which asthenopia is associated with muscular anomalies is not large, and my own experience accords on the whole with this view. Exaggerated asthenopia from

any cause probably does not occur in those whose nervous system is in perfect condition. With regard to the treatment of exaggerated asthenopia, as the underlying cause is always a defective or badly working nervous system, it may be said that it is difficult to correct hereditary influences, and that the patients cannot always change their faulty habits or quickly overcome the effects of mental strain or of the infectious diseases which are so often exciting causes. Anemia, when present, can be benefited by the use of iron, arsenic, or preparations like peptomangan. In cases of exaggerated asthenopia with a relatively acute onset, good results are produced by the removal of such pernicious factors as poor diet or overwork, and by insisting upon proper hygiene, muscular exercise, and, if possible, absolute rest of the eyes. In some instances the persistence of the asthenopia is purely psychical, or, if one chooses to use the term, "hysterical." Among these cases are those in which the asthenopia persists after the tangible physical cause has been removed. Gradle recommends that the patient be persuaded to forget the trouble, and that therapeutic suggestion be used in every manner possible.

Multiple Vision. Multiple vision (*polyopia* or *polyopsia*) is occasionally met with, in some cases the multiple vision being with a single eye and in others with both. It may be of psychical origin, or due conjointly to psychical and retinal conditions. Parinaud ascribes monocular polyopia in hysterical cases to contraction of the ciliary muscle. According to some authorities, it includes *micropsia* and *macropsia*, in the former of which objects at a certain distance from the eye appear smaller than they are in reality, while in the latter when held close to the affected eye they appear enormously magnified, rapidly diminishing in size when removed. Polyopia may also be due to segmental structure of the crystalline lens, or to beginning cataract. Venturi has reported a case of multiple vision in an individual belonging to a family with a strong neurotic tendency. One symptom was a peculiar intolerance of the quinine salts, the use of which was followed in a few hours by pruritus and swelling lasting twenty-four hours and by a troublesome balanitis. The patient became subject to sudden nervous attacks, during which all objects seen were multiplied forty or fifty fold, although consciousness was perfect, and the individual realized that the phenomenon was purely subjective. Venturi inclined to interpret this polyopia as of peripheral origin, due to some spasmodic action of the accommodation muscles. In a discussion of the case, Tamburini held that the multiple vision could also be explained by assuming it to be the result of some special vibratory movement of the perceptive cells in the cortical visual centres, so that there was a lack of fusion of the contemporaneous images. In one case of polyopia with other visual and ocular symptoms, the essential underlying condition was the neuropathic constitution which Gradle has particularly referred to

in describing exaggerated asthenopia, but in addition the patient showed a true delusional tendency. He had carefully worked out his family and personal history, which he always had at ready command. His mother, according to his statements, was born with strabismus, his grandmother at the time of his mother's birth also being treated for her eyes. He was a sickly child until the age of twelve years, and was always troubled with his eyes at night, but could see well enough during the day if he rested his eyes for a time when they began to be tired. Some years before coming under observation he found that the muscles of his eyes, as he expressed it, "would relax overnight," and his eyes become crossed, so that he had double vision in the morning. This double vision could be made to disappear by a strong exercise of will power. Tenotomies were performed on his eyes with some relief, but later he began to be troubled with various other symptoms, among which was polyopia. One day this patient came to me with what he called a "confession," stating that his mind and body had been wrecked by an "overcharge" of belladonna a few years before. This dire result had been brought about by the use of solutions of atropine sulphate, two and four grains to the ounce, three drops of which were instilled three times daily. For ten months after using this solution he remained home in a stupid condition, often seeing before his eyes red lights and phantom images. This case is worthy of record as illustrating the association of two or three causes in the production of a form of asthenopia with diplopia, polyopia, and mental disturbance.

Visual Hallucinations. Visual hallucinations occur chiefly as phenomena of insanity, but are occasionally met with in the sane, in whom they may be due to either peripheral or central irritative disease, usually of a transient character. Occasionally sufferers from general neurasthenia with associated asthenopia have hallucinations of sight. These may be present during states of nervous exhaustion, and at the time between sleeping and waking, or just after the patient assumes a recumbent position. While this subject belongs to the domain of insanity, it may be worth while to cite the methods suggested by Tuke for testing the subjectivity of visual hallucinations, and also the probability of their being either retinal or cortical. Tuke believes that, contrary to what has frequently been stated, careful observation shows that lateral pressure on one eyeball never doubles the subject image. He is also satisfied that with a multiplying lens doubling never occurs without an external object being seen. This makes lateral pressure on the eyeball a trustworthy test of subjectivity. With regard to the differentiation of retinal and cortical hallucinations he calls attention to the fact that in the former the after image of a luminous object obscures or entirely conceals real objects, moves with the motions of the eye, and is "projected" when the observer looks on a dark ground.

CHAPTER VIII.

DISTURBANCES OF OCULAR MOVEMENT DUE TO LESIONS OF THE NERVES, NUCLEI, AND CENTRAL APPARATUS OF THE OCULAR MUSCLES.

ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS RELATING TO THE OCULAR NERVES.

Homologies between the Spinal Nerves and the Cranial Nerves from the Third to the Twelfth inclusive.—The cranial nerves, from the third to the twelfth inclusive, to which, as shown by Hill, the four root theory can be applied, have closer analogies and homologies with the spinal nerves than have the olfactory and optic nerves. Somatic nerves, it will be remembered, are chiefly those distributed to the muscles and the skin, and the splanchnic those which supply the viscera and bloodvessels (pages 8 and 27). These nerves are arranged into a series of four for each spinal segment,—the *ventral motor*, the *lateral motor*, the *visceral*, and the *sensory*. In the spinal cord it is comparatively easy to separate these nerves and their roots into the four sets and to refer them to their anatomical sources, as has been done by Gaskell (see pages 27 and 28). It was long since noted by Sir Charles Bell that the cranial nerves are arranged in at least two linear series. He believed that the lateral group was connected with respiration. According to Hill and Gaskell, through almost the entire length of the spinal cord the ventral, lateral, and visceral roots pass out together by way of the so-called ventral or anterior root bundles, while only the sensory fibres are contained in the posterior gangliated root. Hill and Gaskell gave special attention to the study of the spinal accessory nerve. In the cervical region of the spinal cord the lateral fibres are so separated from the other constituents of the ventral roots as to constitute a true lateral root, which is the origin of the spinal portion of the accessory nerve. In applying the theory of four roots to the cranial nerves the two best guides are the lines of exit of these nerves and the arrangement of their nuclei. The hypoglossal roots arise between the pyramid and the lower olive, and the sixth and third nerves in nearly the same positions with regard to the mesal line. The nuclei of these nerves also lie in an interrupted line, having relatively about the same position to the mesal line; the nucleus of the fourth nerve also belongs to this group, although the mode of emergence of the nerve is different. These four constitute a series of nerves which correspond in their origins with the nerves derived from the ventral or

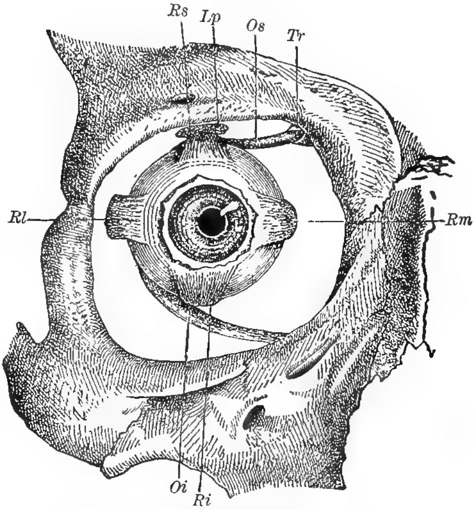
anterior horns of the spinal cord. They are all motor in function. The motor portions of the other cranial nerves are situated more laterally. The spinal portion of the accessorius arises, as already shown, from the lateral horns of the cord. The motor roots of the vagus, glossopharyngeus, and trigeminus, and the facial roots which are entirely motor, arise in more laterally situated gray matter, their nuclei of origin corresponding more closely to the lateral horns of the cord. The eighth or so-called auditory nerve, as already shown, has a lateral or cochlear root and a mesal or vestibular root. In the present chapter the three motor nerves which supply the ocular muscles will be considered together, although in the usual enumeration of the cranial nerves the third and fourth are separated by the trigeminal and the facial nerve from the sixth.

Origin and Course of the Sensory and the Motor Cranial Nerves compared.—The sensory nerves, afferent in type, have their nuclei of origin outside of the neuraxis, in close relation with their peripheral end organs. The origin of the optic nerve has been sought in the retina, of the olfactory nerve in the nasal mucous membrane, of the gustatory nerves in the petrous, jugular, and geniculate ganglia, of the cochlear nerve in the organ of Corti, and of the vestibular nerve in the organ of Scarpa. These nerves have been traced from their points of origin to their terminal nuclei in the brain stem, and in their further course to the cortex. The motor cranial nerves, now to be considered, need to be treated somewhat differently. They are efferent in type, and have their nuclei of origin in the brain stem. The cerebral regions related to them might with propriety be considered first, and indeed these have already been discussed under cerebral localization. After a glance at their cerebrobulbar connections, they will therefore be traced from their nuclei of origin in the brain stem.

The Musculature of the Eye.—The muscles which go to the eye are divisible into those which are distributed to the outside of the eyeball, the *extraocular muscles*, and those which are distributed to structures within the ball, the *intraocular muscles*. The extraocular muscles are seven in number,—the internal straight (*rectus internus*), the superior straight (*rectus superior*), the inferior straight (*rectus inferior*), the external straight (*rectus externus*), the superior oblique (*obliquus superior*), the inferior oblique (*obliquus inferior*), and the elevator of the upper lid (*levator palpebræ superioris*). The intraocular muscles are three in number, namely, the ciliary muscle (*musculus ciliaris, ligamentum ciliare, annulus ciliaris, tensor choroidea, musculus Brueckianus, ganglion ciliare, Bowman's muscle*), the sphincter of the iris (*sphincter iridis, sphincter pupillæ, contractor pupillæ, musculus circularis iridis*), and the dilator of the iris (*levator pupillæ, dilator pupillæ, musculus radialis iridis*). For more than half a century the existence of the pupillary sphincter has not been seriously disputed,

but whether a set of true dilating fibres can be demonstrated in the iris has been doubted by able observers. It is now, however, generally agreed that a radially arranged dilator muscle is present in the iris, microscopical investigations made in recent years having demonstrated the existence in the iris of a layer of muscular fibres probably having dilator functions. The name *Mueller's muscle* or the orbital muscle of Mueller is applied to a peculiar structure formed of radiating muscular bands which pass into or are interwoven with the orbital periosteum as the latter covers the sphenomaxillary fissure. Under certain circumstances it plays a part in the protrusion of the eyeball. The term "Mueller's muscle" is also sometimes applied to the ciliary muscle. It is best, however, to confine this term to the orbital muscle of Mueller. The name Mueller's superior palpebral muscle is sometimes applied to a few unstriated muscular fibres found in the upper lid beneath the true levator palpebræ. This muscle is innervated by the sympathetic, and its paralysis causes a slight ptosis which is usually associated with myosis and retraction of the pupil. The so-called inferior palpebral muscle of Mueller is a corresponding band of smooth muscle fibres inserted at the inferior border of the lower tarsus, in what has been recorded as an extension of the sheath of the rectus inferior.

FIG. 378.

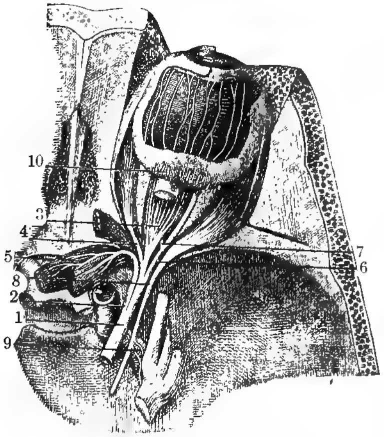


Position of eyeball in the orbit, showing the insertions of the most important intraocular muscles: *Lp*, levator palpebræ superioris; *Rs*, rectus superior; *Rm*, rectus medialis (internal straight); *Rl*, rectus lateralis (external straight); *Os*, obliquus superior; *Tr*, trochlea, or pulley; *Oi*, obliquus inferior; *Ri*, rectus inferior. (Merkel.)

The Nerves of the Ocular Muscles.—The nerves of the ocular muscles are derived from the third, fourth, and sixth pairs, the cavernous plexus of the gangliated (sympathetic) system of nerves, and the nasal branch of the fifth nerve. The third nerve supplies both extraocular muscles (the internal, superior, and inferior straight muscles, the inferior oblique, and the elevator of the upper lid) and intraocular muscles (the ciliary muscle and the sphincter of the pupil). The fourth nerve is distributed solely to the superior oblique muscle, and the sixth nerve solely to the external straight muscle. The radiating fibres of the iris (dilator pupillæ) are supplied by fibres from the cavernous plexus. Nerves of ordinary sensation and

trophic nerves, branches of the nasal subdivision of the fifth nerve, pass to the eyeball, to the muscles as well as to other structures. The sympathetic fibres to the iris, and the branches of the third nerve to the ciliary muscle and to the pupil, are included under the general head of ciliary nerves, these piercing the sclerotic around the optic nerve to go forward between the sclerotic and choroid coats. Numerous other branches of the fifth nerve and of the sympathetic enter the orbit. The motor nerves for the eye—the third, fourth, and sixth—are best considered together, owing to their closely associated functions; but, while they should be discussed in the same connection, a proper explanation of their diseases requires also their separate consideration. Lesions specially located may cause symptoms indicating involvement of the whole or of a portion of only one of these nerves. For the proper understanding of some clinical cases it is especially important to bear in mind the subdivisions of the third nerve. While the consideration of the ciliary (ophthalmic or lenticular) ganglion properly belongs to the fifth nerve, owing to its intimate relation especially with the neural apparatus for ocular movements, it may be well to refer briefly to it in this connection. It is situated about one quarter of an inch in front of the sphenoidal fissure, between the optic nerve and the external straight muscle. It has a motor, a sensory, and a gangliated (sympathetic) root. The motor root, which is derived from that branch of the third nerve which goes to the inferior oblique muscle, enters the caudoventral portion of the ganglion. Six short ciliary nerves arise from the cephalic portion of the ganglion, and these, subdividing and communicating with the long ciliary nerves, form about twenty nerves which separate into a superoexternal and an inferointernal group (Morris).

Fig. 379



Oculomotor and abducent nerves, the upper part of the right orbit removed: 1, third or oculomotor nerve: 2, 3, branches to the superior straight and palpebral elevator muscles; 4, 5, branches to the internal and inferior straight muscles; 6, branch to the inferior oblique muscle; 7, branch from the latter to the ciliary or ophthalmic ganglion; 8, abducent nerve to the external straight muscle; 9, communicating filaments between the abducent nerve and the carotid plexus of the sympathetic (to their outer side is the trifacial nerve); 10, ciliary nerves perforating the sclerotica and passing forward between it and the choroidea to the ciliary muscle and iris. (After Hirschfeld-Sappey.)

Connections of the Optic Nerve with the Nidi of the Nerves of the Ocular Muscles.—Certain afferent fasciculi of the optic nerve pass to the pregeminum, in the nuclei of which are cells of large size. Axis cylinders pass from these cell bodies, and probably some con-

stituent fibres of the dorsal longitudinal bundle. They give off collaterals which ramify among the root fibres of the third, fourth, and sixth nerves, and thus is established a communication between the optic nerve fibres and the cell nests of all the motor nerves of the muscles of the eye. A part of the fibres of the posterior longitudinal fasciculus, however, probably comes from the gray matter surrounding the third ventricle.

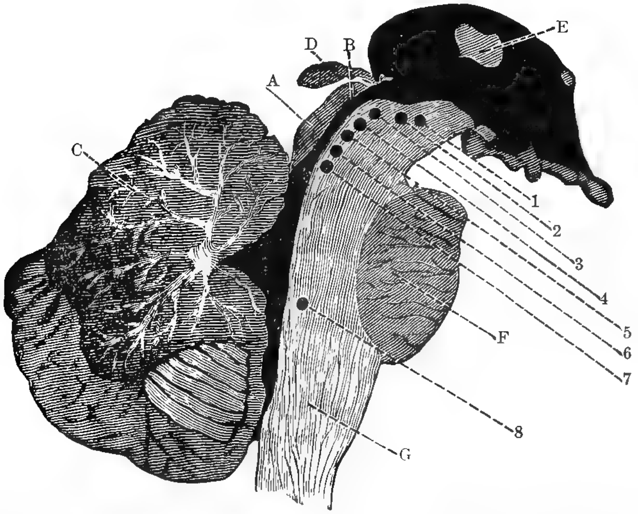
The Oculomotor Neurons from the Cortex to the Periphery.—The central or cortical oculomotor neurons originate in a locality which, as already discussed (cerebral localization, pages 337–339), has not yet been definitively settled. Originating in the cells of either the motor or the visual cortex, the axis cylinders pass about the knee of the internal capsule probably anterior to the fasciculus of the pyramidal tract, the bundle containing the fibres of these oculomotor neurons being sometimes called Spitzka's bundle. In general terms this bundle may be described as probably situated in the mesal portion of the pyramidal region in the peduncle, here turning mesad and dorsad to terminate in the nuclei of the motor oculi of the opposite and probably of the same side. Around the ganglion cells of these nuclei are found the terminal ramifications of these central oculomotor neurons. In these cells and their arborizations begin the peripheral neurons, the nerve fibres passing out of the nuclei as the roots of the motor oculi nerves on the same side, and also in lesser part decussating with fibres of the other side.

End Organs of the Oculomotor Nerves.—Motorial end plates (see page 18) are found in the ocular muscles as in those of other parts of the body. The short ciliary nerves, derived from the ciliary ganglion, are probably of mixed function. After entering the ciliary muscle, they unite to form the ciliary plexus (*plexus gangliosus ciliaris, orbiculus gangliosus ciliaris*). From this plexus fibres pass to the iris, the ciliary muscle, and the cornea. Special motor endings and a special apparatus related to the muscular sense have been recently described as present in the ciliary body.

The Nuclei of the Third Nerve.—*General Historical Summary.* Formerly, as by Meynert and his followers, the oculomotor or third nerve was believed, like other motor cranial nerves, to have a common, single, and undifferentiated nucleus or cell nest on each side of the mesal line. Gradually one nucleus after another has been differentiated. Hensen and Völckers (1878) believed that they had located in rabbits the nuclei governing the various functions of the third nerve in the following order from before backward: ciliary muscle (accommodation), sphincter iridis (light reflex), rectus internus, rectus superior, levator palpebræ, rectus inferior, obliquus inferior. Von Gudden (1880) found two nidi for each oculomotor nerve, one decussating with another cell nest on the opposite side of the mesal line, the other lateral and giving origin to those fibres of the third

pair which remain on the same side. Spitzka (1885) confirmed these views, finding that the innermost and most posterior rootlets cross the raphe. Kahler and Pick (1881), while admitting that the centres for the nerve supply to the sphincter pupillæ and ciliary muscles lie detached from those for the fibres passing to the external muscles, also supplied by the third nerve, were led from the postmortem examination in two cases of incomplete nuclear paralysis of the third nerve to locate the nuclei of the nerves to the levator muscles in a line lying to the outer side of those destined for the supply of the rectus internus and rectus inferior. This brings

FIG. 380.



Longitudinal vertical section through the human brain, showing (diagrammatically) the position of the nerve nuclei of the ocular muscles, according to Hensen and Völckers: A, postgeminum; B, pregeminum; C, cerebellum; D, conarium; E, gray commissure in the middle of the third ventricle, which, with the iter and fourth ventricle, is represented in black; F, pons; G, postoblongata; 1 to 6, different centres of the third nerve nucleus, viz., 1, for accommodation; 2, for the sphincter of the pupil; 3, for the rectus internus; 4, for the rectus superior; 5, for the levator palpebræ superioris; 6, for the rectus inferior; 7, nucleus of the fourth nerve (trochlearis) for the superior oblique; 8, nucleus of the sixth nerve for the rectus externus. (Bramwell.)

the centres physiologically associated close together. Westphal (1888) sharply demarcated a subnucleus of the oculomotor nerve, cephalad of the main nucleus, this according to him and Edinger (1885) being connected with the innervation of the interior muscles of the eye (with the ciliary or the iris muscle or with both). Spitzka (1888) found in reptiles but little differentiation of the cell nests of the third pair; in birds he found that the nucleus of the third pair is very large; that in the dog, cat, sea-lion, and lion, the nuclei were disposed in a peculiar manner much as in man; that in crepuscular bats the cell nests were very small; while in the mole they were almost absent, and only in anthropoid apes was the anatomy of the cell

nests and their intranidal tracts approximately as complex as in man. Knies (1894) suggests that the Edinger-Westphal centre is for accommodation only, and the Darkschewitsch centre for the regulation of pupillary movements. Mendel (1887) located the centre for the oculofacial movements at the posterior extremity of the oculomotor nucleus, although this localization is not usually accepted. The centre for the levator palpebræ has been placed by me just posterior to the centre for convergence, as shown in the scheme Fig. 383. Darkschewitsch (1889) located a cell nest in the upper part of the aqueduct above the third nucleus and extending into the walls of the third ventricle, the cells having no connection with the main oculomotor nucleus, but being connected with the postcommissure and the posterior longitudinal fasciculus. Siemerling (1891) in a study of a case of unilateral congenital ptosis observed degenerative changes in the chief oculomotor nucleus, these being bilateral and in the proximal portion. The ventral and dorsal nuclei were equally affected. In the paper in which he contributed this observation he argued in favor of an anterior position for the nidus of the levator palpebræ superioris. Obersteiner speaks of a lateral nucleus which sometimes may be divided into two parts, a medioventral and a dorsolateral nucleus, some of the cells of which are found in the posterior longitudinal fasciculus, or even ventrad to this.

Perlia's Central Nucleus. Commonly the muscles of accommodation and convergence (ciliary and internal straight muscles) act together, as in accommodation for near vision, but this conjoint action is not invariable. A separate convergence centre therefore doubtless exists, and this, it will be readily understood, should be situated in the median line. Such a centre has been described by Perlia (1889), and is sometimes designated as *Perlia's central nucleus.* It is composed of ganglion cells from both sides, meeting in the mesal line beneath the iter. This convergence centre is distinct from, although closely related both anatomically and physiologically to, the centres for the internal straight muscles, which are situated on each side of the mesal line. It is a centre for the associated internal movement of both eyes.

Bruce's Investigations. Bruce (1893) found a series of separate nuclei the whole or a portion of which can be related to the separate oculomotor centres. He found a comparatively long anterior or ventral group of cells close to and a little removed from the median line. In this long anterior or ventral group of cells are probably included several of the centres as given by Perlia and Knies, those, for instance, for the inferior oblique, rectus internus, and rectus inferior. He describes also a posteroexternal group of cells, probably corresponding to the centres for the inferior oblique (dorsal nucleus of Edinger and Siemerling), and in his description is included an additional posteromesal or pale nucleus, which probably corresponds

to the Edinger-Westphal nucleus. Bruce's superior nucleus corresponds to the anterolateral or small-celled nucleus of Darkschewitsch, the centre for the sphincter pupillæ. A small external nucleus described by Bruce has not been, so far as I know, correlated with any special nerve distribution.

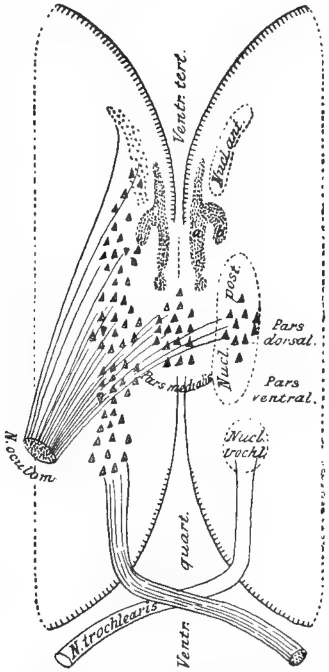
Researches and Views of Koelliker, Starr, Bernheimer, and others. According to Koelliker, the third nerve arises in a nucleus about five millimetres long; according to Perlia, it is about ten millimetres, but he includes Darkschewitsch's nucleus, which Koelliker does not. Koelliker thinks that a sharp separation of the nuclei of the third nerve in man is scarcely possible. He found that the third nerve has essentially only one nucleus in embryos, at the cerebral end of which a round nucleus branches off. He holds to only one chief nucleus with two subdivisions, the dorsolateral with large cells and the dorsomesal with smaller cells, although he admits a central nucleus, with large cells in the proximal portion of the third nucleus, and many nests of cells within the dorsal longitudinal fasciculus. A connection of the third nucleus with the pyramidal tracts has not been positively shown, although it probably exists. The connection of the dorsal longitudinal fasciculus with the third, fourth, and sixth nerves is quite certain. Koelliker was the first who showed that the fibres of the posterior longitudinal fasciculus give off numerous collaterals, and this finding has been confirmed. These collaterals end in the nucleus of the twelfth and in the nuclei of the three eye nerves. It is impossible in the scope of a chapter like the present to refer even briefly to all the numerous workers who have contributed to our knowledge of nuclear anatomy and physiology and to our means of applying this knowledge. Starr was one of the first to construct a table locating the relative positions of the groups of cells governing the ocular muscles, his conclusions being based upon a careful, critical analysis of a large number of cases compiled from the literature of the subject. Edsall and Diller have contributed a paper based upon the report of an interesting case exhibiting bilateral palsy of the superior rectus muscle, the iris, and the ciliary muscle, with loss of power of convergence, and have constructed a tabular arrangement of the groups of cells in the anterior portion of the general nucleus for the different constituents of the third nerve, as follows:

Ciliary muscle.
Convergence.
Sphincter iridis.
Rectus superior.
Levator palpebræ, rectus internus.
Rectus inferior, obliquus inferior.
Obliquus superior, fourth nerve.

One of the most recent and authoritative investigations is that of Bernheimer (1897), who extirpated certain ocular muscles and then

examined the nuclei of the third nerve by the method of Nissl. He operated on rabbits and monkeys. In the latter the third nerve centres seem to be arranged in the same order as in man. He destroyed all the external ocular muscles except the levator palpebræ. According to him, the centres for the external ocular muscles innervated by the third nerve are in the distal and middle thirds of the lateral chief nucleus and in the "lateral cells" (groups of cells between and on the outer side of the dorsal longitudinal bundles). In the more distal portion of the centres the nuclei are chiefly on the side opposite to the nerve; in the middle third they are nearly equally distributed on both sides of the raphe. According to him, the most anterior part of the lateral chief nucleus, the double small-celled Edinger-Westphal nucleus, and the large-celled median nucleus are respectively the centres for the levator palpebræ, the iris, and the ciliary muscle. The Edinger-Westphal nucleus of the right side supplies the right eye; the large-celled median nucleus supplies both eyes. From a comparison of the different facts and views thus outlined, and in accordance with our own present experience, we have arrived at conclusions with regard to the arrangement of the nuclei of the nerves of ocular movements and of their central and peripheral tracts as exhibited in the scheme Fig. 383, page 808.

FIG. 381.



A partly diagrammatic view of the floor of the aqueduct, looking upward (dorsally): nuclei of the third and fourth nerves and the decussating fibres of the latter all shown; the third nerve nuclei are subdivided into an anterior nucleus, the Edinger-Westphal nucleus (*a* and *b*), and a posterior nucleus; the posterior nucleus has a dorsal, a ventral, and a mesal portion; the decussation of the fibres from the dorsal portion of the posterior nucleus of the third nerve is shown. (Edinger.)

General Course of the Third or Common Oculomotor Nerve.—Although the third pair of cranial nerves is commonly designated as the oculomotor (*motor oculi*), this name is not a good one, as the fourth and sixth nerves are also concerned with ocular movements, and all three sets of nerves can be more properly spoken of as "oculomotor." It must, however, always be remembered that in common usage the term oculomotor or motor oculi is applied only to the third nerve. Emerging from the inner side of the crus, close to the upper border of the pons, the numerous root fibres of this nerve combine into one bundle. The nerve trunk thus formed passes between the supercerebellar and postcerebral arteries, forward and

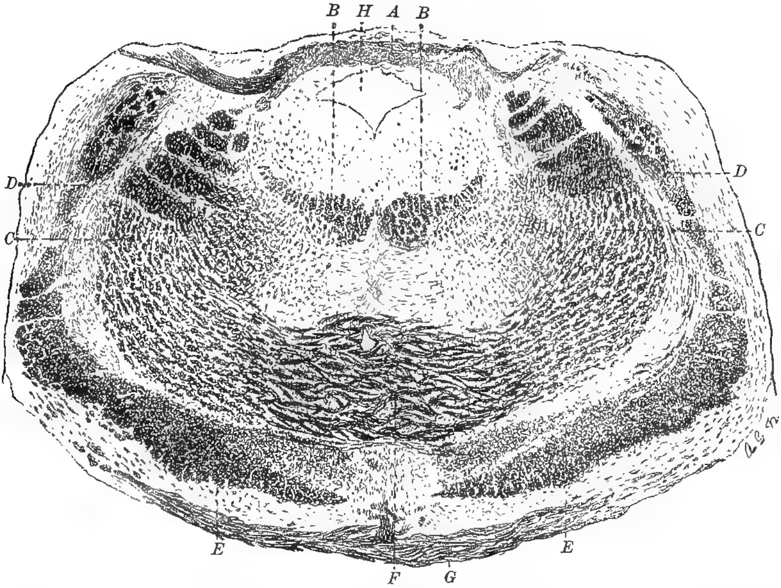
slightly outward, soon to pierce the dura between the anterior and posterior clinoid processes. It then runs forward in the outer wall of the cavernous sinus to the sphenoidal fissure, where it is above and internal to the fourth nerve. It here subdivides and sends branches to the various extraocular and intraocular muscles, which it supplies. While in the outer wall of the cavernous sinus it receives slender branches from the cavernous plexus, and also a minute branch from the ophthalmic division of the fifth. It has superior and inferior subdivisions, branches passing from the former to the rectus superior and the levator palpebræ, and from the latter to the rectus internus, rectus inferior, and obliquus inferior. The particular branch which goes to the last sends an offshoot to the ciliary ganglion. This branch, as already stated, contains fibres which eventually supply the ciliary muscle and the sphincter of the pupil.

Structures adjoining and closely connected with the Third Nerve.—The third nerve in its course after its emergence from the crus, "superficial origin," on its way to the orbit, lies adjacent to a number of important structures. These, and the symptoms resulting from lesions affecting them, have been referred to when considering cavernous sinus thrombosis (page 307). At the position of the cavernous sinus the third and fourth nerves and the ophthalmic branch of the fifth lie close to each other, in the order given from above downward and from within outward. The sixth is separate, being close to the carotid artery in the cavernous sinus, and lying internal to the ophthalmic nerve. The somewhat separated position of this nerve accounts for its escape in some cases of basal lesion in which the third, fourth, and ophthalmic division of the fifth are implicated. Near the sphenoidal fissure the sixth nerve is close to the others. In the fissure the fourth and the frontal and lachrymal branches of the fifth lie on the same level, the fourth being the nearest to the inner side. They enter the orbit in the following order from above downward: the upper division of the third, the nasal branch of the fifth, the lower division of the third, and, lowest of all, the sixth. (Quain's Anatomy.)

The Nucleus and the Root Fibres of the Fourth Nerve.—The position of the nucleus of the fourth nerve is shown in several illustrations (Figs. 6, 88, 381, and 388). It appears practically as a caudal continuation of the series of nuclei of the third nerve, from which it is not clearly separated. It is dorsal to the dorsal longitudinal bundle and occupies a concavity in this fasciculus, and is revealed by a transection through the cephalic extremity of the postgeminum. Its root fibres have a considerable course within the brain stem, emerging just caudad of the postgeminum. As Obersteiner observes, nothing is more certain in brain anatomy than the crossing in the valvula of the vast majority of root fibres which constitute the fourth or trochlear nerve. Some of the root fibres

of this nerve, however, probably emerge on the side of origin, in this respect following the same rule as the other motor cranial nerves. It has connections with the cerebrum, the pregeminum, and the dorsal longitudinal bundle, similar to those of the third and sixth nerves. According to some authorities, it has crossed relations with the nucleus of the abducens. Axis cylinders pass from the nerve cells of the cortex to the nucleus of origin of the fourth nerve, constituting the first neuron of this nerve series. A group of small cells caudad to the fourth nucleus was supposed by Westphal to belong to this nucleus. This is probably incorrect.

FIG. 382.



Microscopical section through the decussation of the fourth nerve: *A*, decussation of the fourth nerve; *B*, *B*, dorsal longitudinal bundles; *C*, *C*, prepeduncles; *D*, *D*, lateral (lower) lemnisci; *E*, *E*, mesal (upper) lemnisci; *F*, decussation of prepeduncles; *G*, deep transverse fibres of the pons; *H*, iter. Between the iter and the most lateral portion of the dorsal longitudinal bundles the large cells of the locus ceruleus are seen.

Peripheral Course of the Fourth Nerve.—The fourth (pathetic or trochlear) nerve trunk emerges from the valve of Vieussens near the frenulum (cephalic border of the valvula), just below the postgeminum, and, crossing the prepeduncle, winds around the crus between the supercerebellar and postcerebral arteries, reaching the ventral aspect of the pons at its cephalic margin. It pierces the dura near the posterior clinoid process, and passes cephalad in the wall of the cavernous sinus, lying between the third nerve, which is above and internal to it, and the ophthalmic branch of the fifth nerve, below and external to it. When it reaches the sphenoidal fissure it bends upward, crosses the third nerve, and, passing into

the orbit, above the external straight muscle, and also over the levator palpebræ and superior straight, ends in the superior oblique muscle. This nerve is the smallest in diameter of the cranial nerves, and it is distributed to a single muscle. In the cavernous sinus it is connected by small filaments both with the sympathetic and with the ophthalmic branch of the fifth. It has been estimated to contain about twelve hundred fibres. Gaskell believes that he discovered near its superficial origin vestiges of a degenerated ganglion.

The Nucleus and the Root Fibres of the Abducens Nerve.—The position of the abducens nucleus, which is almost globular in general shape, is shown in several illustrations (Figs. 6, 84, 383, and 396). According to Obersteiner, the separate bundles of the abducens nerve, traced dorsad in gently curving arches, apply themselves to the mesal side of the nucleus, curve around it dorsally, extending in some cases as far as its lateral aspect, and sink successively into its substance, to their cells of origin. A very small and easily overlooked portion of the abducens turns mesad beneath the nucleus, extends to the raphe, traverses it apparently as far as its dorsal edge, and then, passing beneath the ascending crus of the facial nerve, enters the abducens nucleus of the opposite side. Koelliker has not been able to see the obscure fibres described by Obersteiner as crossing the raphe to the opposite sixth nucleus. Duval has described in the monkey fibres which arise in the sixth nucleus and enter the dorsal longitudinal fasciculus and then pass to the fourth and third nuclei of the opposite side. According to this, the right sixth nucleus would innervate the right rectus externus and the left superior oblique and certain muscles supplied by the third, as, for example, the internal rectus (Duval and Laborde). The central neuron of the abducens passes from the cortex to the abducens nucleus of the opposite side, and the peripheral neuron beginning in this nucleus takes its course, as described when tracing the root fibres and peripheral course of the nerve, to the external rectus muscle. It is customary to consider the fibres connecting the sixth nucleus with the superior olive as part of the central tract of the eighth nerve, which is united by the trapezoid body to the superior olive, and thus to explain reflex movements of the eyeballs due to sound impulses, but the accuracy of this view must be determined by further investigations. It seems to Koelliker just as probable that this connection of the superior olive with the sixth nucleus is a sensory tract of the second order, which by means of the lateral fillet connects the sixth nucleus with the proximal quadrigeminal body and in this way with the optic apparatus. Gudden and Gowers have conclusively shown that the seventh nerve receives no fibres from the sixth nucleus, though at one time the opposite view was held by many. The cerebral connection with the sixth nucleus is probably through the pyramidal tract.

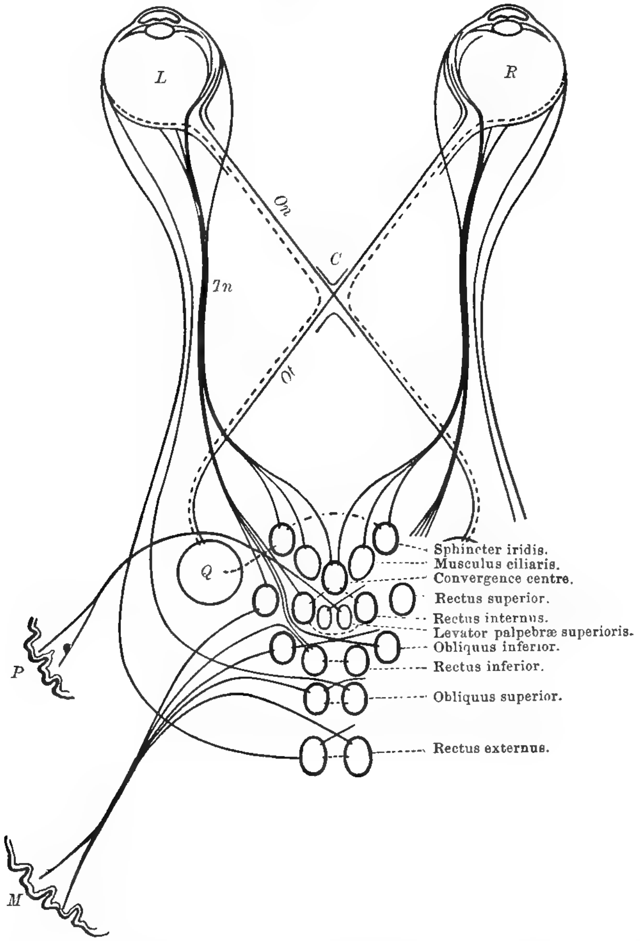
Peripheral Course of the Abducens Nerve.—The root fibres which eventually constitute the trunk of the abducens nerve emerge in the furrow between the pons and the postoblongata, immediately external to the pyramids ("superficial origin"). Occasionally one or two of the fibres are distinct from the others at their place of emergence. The rounded nerve trunk passes forward along the ventral surface of the pons, and pierces the dura to the inside of and below the fifth nerve. It crosses the apex of the petrous bone, enters the wall of the cavernous sinus, and, proceeding forward internally along the outer wall of the carotid artery, passes through the sphenoidal fissure into the orbit, to be distributed to the rectus externus muscle. It is below the other nerves as it enters the orbit. It receives small filaments from the cavernous plexus and from the ophthalmic division of the fifth nerve. It has been estimated to have as many as three thousand fibres in its trunk.

Scheme of the Nuclei for Ocular Movements.—The scheme shown in the diagram Fig. 383 seems to us most in accord with the most recent facts relating to the oculomotor mechanism; but it must be remembered that the relative positions of some of the centres illustrated in this diagram have not been conclusively fixed. This remark is especially applicable to the location assigned to the centres of the levator palpebræ, which according to some authorities are the most anterior of the centres for the ocular nerves. According to this diagram, the most cephalic or anterior centres are those for the ciliary muscle and for convergence, the centres for the levator palpebræ being placed close to the mesal line caudad of the convergence centre. These centres are situated in the central cinerea, close to the caudal extremity of the third ventricle, and therefore close also to the anterior extremity of the iter. The centres for the other ocular muscles are given in what we believe to be the order which corresponds with the facts as generally accepted, as follows: rectus superior, rectus internus, obliquus inferior, rectus inferior, obliquus superior, and rectus internus. The decussations of the root fibres of certain of these centres, their connections with the pregeminum and the optic tract, and their relations to the cortex are also shown in the diagram.

The Ciliospinal Centre.—Numerous observations, physiological and clinicopathological, have corroborated the existence and position of a ciliospinal centre extending from the sixth or seventh segment of the cervical portion of the spinal cord downward to the region from the first to the third thoracic segment. Unilateral extirpation of the cord in this region causes myosis and irritation mydriasis or dilatation of the pupil on the same side. Fibres pass from this area through the ventral roots of the two lowermost cervical and two uppermost thoracic nerves into the cervical gangliated (sympathetic) system of nerves. In goats and cats this centre, even after separation from the oblongata, can be excited directly by dyspneic blood,

and also reflexly by the stimulation of sensory nerves, especially when the reflex excitability of the cord is increased by the action of strychnine or atropine. Certain radiating smooth fibres in the eye-

FIG. 383.



Scheme of the nuclei of the nerves of ocular movement and of their central and peripheral tracts: *R*, right eye; *L*, left eye; *C*, chiasm; *On*, optic nerve; *Ot*, optic tract; *Q*, pregeminum (anterior quadrigeminal body); *P*, cortical centre for the movement of elevation of the upper eyelid; *M*, cortical centre for ocular movements; *Tn*, course of all the ocular nerves in the cavernous sinus. The names of the different nuclei are printed on the diagram, and the nerve tracts going from these nuclei can be readily traced to where they converge in their course in the cavernous sinus and where they diverge to pass to the various muscles of the eye. The dotted lines represent associating and commissural tracts.

lids, the dilator fibres of the iris, and the so-called Mueller's muscle in the orbit, it is believed, are supplied from the ciliospinal centre. The fact that elevation of the upper eyelid, dilatation of the pupil, and protrusion forward of the eyeball are ocular phenomena which occur together, affords a clinical illustration that all these movements

are probably controlled from a single central region. Since the investigations of Madame Dejerine-Klumpke we have a form of paralysis known by her name, due to a lesion of the lower roots of the brachial plexus, *i.e.*, of the eighth cervical and first thoracic root fibres. In this form of paralysis the small muscles of the hand, and some of the muscles of the forearm, especially the flexors, are involved. Sensation may be altered in the distribution of the ulnar and median nerves and on the inner side of the forearm. If these roots of the brachial plexus are affected in the portions which contain the fibres of the rami communicantes, oculopupillary symptoms are usually present. Madame Dejerine-Klumpke demonstrated that the oculo-pupillary fibres in the dog leave the spinal cord by means of the first thoracic roots, and since the publication of her experiments numerous cases have shown that these fibres in man have the same course, but it is not positively known whether the eighth cervical and second thoracic roots also contain some of these fibres.

Physiological Action of the Ocular Muscles.—The lateral movements of the eye are regulated by the rectus internus and rectus externus; the upward movements, by the rectus superior and obliquus inferior; the downward movements, by the rectus inferior and obliquus superior; the movements upward and inward, by the superior and internal straight acting with the inferior oblique; the downward and inward, by the inferior and internal straight acting with the superior oblique; upward and outward movements, by the superior and external straight acting with the inferior oblique; and downward and outward movements, by the inferior and external straight acting with the superior oblique. The clinician is chiefly concerned with the lateral and vertical movements, although movements in special directions may have a particular interest in rare cases. The excursion of the eyes in the vertical and horizontal directions amounts to about ninety degrees. (De Schweinitz.)

DISTURBANCES OF OCULAR MOVEMENTS.

Terminology and Symptomatology.—*Miscellaneous Terms.* Certain terms relating to the oculomotor apparatus have already been defined, as nystagmus and exophthalmus (page 159), iridoplegia, hippus, and other pupillary phenomena (pages 162 and 163), and conjugate deviation (page 166). Some of the terms are so well known as hardly to need explanation. *Monocular vision* is vision with one eye, and *binocular vision* is the vision which results from using both eyes together without the production of diplopia. Stereoscopic vision is a form of binocular vision. *Diplopia*, or double vision, is usually binocular, and then depends upon the unequal or unbalanced action of both eyes; but it may be *monocular*, resulting from the action of only one eye, in which case it is commonly dependent upon some imperfection of the iris or of the ocular media.

False projection is a symptom produced when the position of an object cannot be accurately estimated by vision. Normally objects are so projected with regard to vision that the observer can accurately estimate the position in the visual field, but some forms of paralysis and of paresis of the ocular muscles cause false projection. *Mydriasis*, the term applied to dilatation of the pupil, is divided into a *paralytic* and an *irritation* variety. The former is dependent upon destruction of the centre for pupillary contraction, or of the fibres which go to or from this centre, or is due to lack of light stimulation from the retina. The latter results from irritative lesions. Contraction of the pupil known as *myosis* may also be of the *paralytic* or of the *irritation* variety, the former being due to a destructive lesion causing paralysis of the centres or fibres which control the dilatation of the pupil, and the latter to irritation of the same centres or fibres. Special forms of mydriasis or myosis according to the locality of the lesion are sometimes described: thus we may have a spinal myosis or a cerebral mydriasis.

Paralyses and Insufficiencies of Ocular Muscles. From some affections of the oculomotor apparatus result various forms of *strabismus*, that abnormality of the eyes and vision in which the visual axes do not meet at the point at which they would normally come together if the actions of the external ocular muscles were properly coordinated. In recent years various convenient terms expressive of the position of the visual axes in partial or complete strabismus have been introduced into ophthalmological and neurological nomenclature. The general term *heterotropia* is used as descriptive of deviation of any kind; *hypertropia*, when one visual line is placed above another; *esotropia*, for deviation inward; and *exotropia*, for deviation outward. In like manner *heterophoria* is used to indicate the tendency of visual lines to take some other direction than that of parallelism, and according as the direction is upward, inward, or outward the effect is described as *hyperphoria*, *esophoria*, or *exophoria*. *Hyper esophoria* expresses the tendency of the visual lines to be directed both upward and inward, while *hyperexophoria* indicates a tendency both upward and outward. These affections, which result from insufficient or incoordinate action of the ocular muscles, are in some instances dependent upon conditions which properly claim the attention of the neurologist. They are often classed under the general head of *muscular asthenopia*.

Forms of Ocular Deviation. As the result of paralysis of certain of the ocular muscles, various forms of deviation may occur. The term *primary deviation* is applied to those cases in which the direction taken is away from the muscle affected. The limitation is in the direction of what would be the action of the affected muscle. In *secondary deviation* the affected eye fixes while the normal eye deviates. The amount of stimulus required for the affected eye is such

as abnormally to stimulate the normal eye and thus bring about this deviation.

Ophthalmoplegias. The term ophthalmoplegia is frequently and somewhat loosely used in describing various forms of ocular paresis or paralyses. It means simply paralysis of the muscles concerned in ocular movements, and is sometimes employed in this broad sense, but it has been more generally applied to particular forms of progressive and more or less symmetrical paralysis of the muscles of the eyeballs. It is true, as has been said by Jeffries, that to make a diagnosis of "ophthalmoplegia" is about as significant as to make that of stomachache, but nevertheless it will be found convenient to define and consider separately some forms of ophthalmoplegia. *External ophthalmoplegia*, rare as an isolated affection, is paralysis of the extraocular muscles. The term *internal ophthalmoplegia* is applied to paralysis both of the ciliary muscle (*cycloplegia*) and of the sphincter of the iris (*iridoplegia*). In cycloplegia the pupil does not respond to efforts at accommodation. Iridoplegia shows itself by dilatation of the pupil, and may be of several varieties. In *reflex iridoplegia*, commonly called the *Argyll-Robertson pupil*, usually the pupil is small, or it may be of normal size, or even dilated, the essential factor being the nonresponse of the iris to the stimulus of light. Accommodation may be affected in iridoplegia, but usually it is not. It may be either unilateral or bilateral, and occasionally curious combinations or dissociations of iridoplegia and cycloplegia are observed. Schwarz, for instance, has reported a case of right incomplete reflex iridoplegia and left incomplete accommodation palsy. In rare cases the converse of the Argyll-Robertson pupil is observed, the pupil or pupils responding to light, but not contracting during efforts at accommodation and convergence. No special term has, so far as I know, been applied to this particular form of internal ophthalmoplegia. Ophthalmoplegias are sometimes subdivided and named according to the locality of the lesion producing them; thus they may be *cortical*, *subcortical*, *peduncular*, *nuclear*, *radicular*, *commissural*, *trunkal*, *orbital*, and *endocular*. The general term *basal* is sometimes applied to different forms of ophthalmoplegia due to lesions variously situated at the base of the brain. Ophthalmoplegias have been designated by some authorities as *fascicular*, this term simply referring to the fact that nerve bundles and not nerve centres are affected by the lesions. Each of the above anatomical or topographical forms of ophthalmoplegia will receive attention in its proper place.

Ophthalmospasms. Not a few of the affections of the intraocular muscles are spasmodic rather than paralytic, hyperkineses and not akineses. To these the term ophthalmospasm may be conveniently applied. Such spasms may be *clonic* or *tonic*. They may also be either *functional* or *organic*, and functional cases may be further

subdivided into *hysterical* and *neurasthenic* forms. Special varieties of ocular spasm are also spoken of, according to the muscles affected, as *blepharospasm*, or spasmodic closure of the lids, due chiefly to spasm of the orbicularis palpebrarum ; *ciliary spasm*, when the ciliary muscle is affected ; *spasmodic ptosis*, when the spasm is of the levator palpebræ, or of this muscle together with the frontalis or corrugator supercilii ; and *spasmodic strabismus*, which may be of different forms according to the muscles affected. Some of the forms of conjugate deviation of the eyes are spasmodic.

Nystagmus. Nystagmus has already been defined (page 159), and reference has been made to its occurrence and cause in several places (pages 380 and 736). The different varieties of nystagmus are enumerated by Fuchs as *nystagmus oscillatorius*, oscillating or vibrating nystagmus, *nystagmus rotatorius*, or rolling nystagmus, and *nystagmus mixtus*, in which the oscillatory and rotatory movements are combined in the same case. Nystagmus is usually bilateral, but may be more marked in one eye than in the other, and even in rare cases may be present in only one eye. It may be caused by amblyopia, blennorrhœa, corneal opacities, and absence of choroidal pigment. As has been indicated in several cases, it is a not infrequent accompaniment of degenerative diseases, and especially of disseminated sclerosis. The fact in which we are most interested in this connection is, however, that nystagmus may accompany various forms of paralytic and spasmodic disorder of ocular movement, and may be due to lesions either of the nuclei of the ocular nerves or of the associated or commissural tracts which relate these nuclei to each other and to various portions of the brain. In connection with the known facts regarding the association of the cochlear centres with the abducens nucleus it is interesting to note the occasional occurrence of nystagmus in those who have affections of hearing of either peripheral or central origin. Nystagmus has, for instance, been observed in connection with otitis media, in Ménière's disease, and in disease of the auditory centres and tracts. The interrelationships of vertigo and ocular disturbances, including nystagmus, have been discussed under vestibular disease (page 736). Some interesting forms of nystagmus associated with disorders of ocular movement have fallen under my observation.

Methods of Examination.—For minute details regarding the examination of patients suffering from affections of ocular movements, works on ophthalmology should be consulted.* The patient is seated several metres from the testing object, usually a candle flame. It is necessary to have a pair of spectacle frames in which to

* For some of the details given, as well as for other important facts and views, use has been made of the paper on "Eye Paralyses" by Dr. John Amory Jeffries, edited since his death by Dr. Philip Coombs Knapp.

place one colorless and one plain glass. The existence of monocular diplopia is excluded by testing first with one eye and then with the other. The testing flame is moved to the right and left, upward and downward, and in various other directions, the relative positions and the distance between the double images being noted. The fact that one image is colored and the other not makes the procedure easy. When vision is impaired, care must be taken not to mistake monocular vision due to lack of sight in the peripheral portion of one retina for parallel vision. The absence of double vision does not prove the existence of paralysis, as one of the images may be disregarded or lost, or only one image may be present, as in some cases of conjugate paralysis. After testing with the double images the motions of the eyes should be observed when following some object. The range of motion—the excursion of the eyeball—should be noted, as should also the position of the upper lids, and whether they follow the pupils in looking downward. Any differences in the motion of the eye when its mate is covered should be noted, as a paresis or paralysis of one eye may disappear if the other be covered, and in other cases the paralysis is apparent only when the unaffected eye is closed. Secondary deviation of the sound eye is determined by placing a screen between the eye to be tested and the object looked at. When now the screen is removed, if the deviation exists the eye will turn back a few degrees as the object comes into view. The screen should be held sufficiently near to prevent fixation of the eye, and yet so that it can be seen by the observer. In studying false projection, if a small object is fixed with the paretic eye and the patient makes an effort to pick it up quickly the hand will miss the object and go too far in the direction towards which the eye movement is weak. In making this test the other eye should be closed, the motion should be made rapidly, and the object—as a pin stuck into a table—should be so placed as to involve the use of the affected muscles. Besides these tests, which are chiefly for phenomena due to affections of extraocular muscles, the patient should be tested for reaction of the pupil to light (pages 160, 163); he should also be examined for near vision, and sensations of the skin should be noted, as well as the actions of the ciliary muscles.

Ocular Paralysis as studied by the Diplopia Tests.—*Forms of Diplopia.* That form of diplopia showing two horizontal images is called *lateral diplopia.* In *vertical diplopia* the images show a vertical displacement: one is seen above the other. In *simple, direct,* or *homonymous diplopia* each image is seen by the eye of the side on which it appears; that on the right side belongs to the right eye, that on the left to the left eye. In *crossed* or *heteronymous diplopia* each image is seen by the eye of the side opposite to that on which it appears,—that on the right by the left eye, that on the left by the right eye. Diplopia may be either homonymous or heteronymous

in forms of both lateral and vertical diplopia. In the latter, while one image is above the other, it is always displaced a little to the right or to the left. Homonymous diplopia is always associated with convergence, and crossed diplopia with divergence, as shown in the diagram Fig. 384. *Lateral diplopia indicates paralysis of the rectus internus or rectus externus.* When the images are homonymous the paralysis is of the externus, images separating to the right indicating paralysis of the right externus, and images separating to the left paralysis of the left externus. Crossed or heteronymous images indicate paralysis of the rectus internus. Images separating to the right indicate paralysis of the left internus, and images separating to the left paralysis of the right internus. *Vertical diplopia in the upper field indicates paralysis of either the rectus superior or the obliquus inferior.* Like lateral diplopia, it presents itself in two forms, one homonymous and the other heteronymous. Homonymous images indicate paralysis of the obliquus inferior, and when the image of the right eye is higher than that of the left it means paralysis of the inferior oblique of the right eye, while if the image of the right eye is lower than that of the left it means paralysis of the inferior oblique of the left eye. Crossed images in vertical diplopia in the upper field indicate paralysis of the superior rectus. When the image of the right eye is higher than that of the left the paralysis is of the rectus superior of the right eye, while when the image of the right eye is lower than that of the left the paralysis is of the rectus superior of the left eye. *Vertical diplopia in the lower field indicates paralysis of the rectus inferior or obliquus superior.* Homonymous images indicate paralysis of the superior oblique. If the image of the right eye is lower than that of the left the paralysis is of the superior oblique of the right eye, and if the image of the right eye is higher than that of the left the paralysis is of the superior oblique of the left eye. Crossed images indicate paralysis of the rectus inferior, the image of the right eye being lower than that of the left in paralysis of the rectus inferior of the right eye, and the image of the right eye being higher than that of the left in paralysis of the rectus inferior of the left eye. (Hotz.)

FIG. 384.

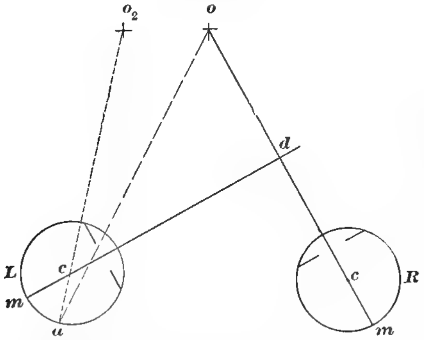


Diagram showing the difference between homonymous and heteronymous images: o, object fixed by the right eye, R; L, left eye; mcd, visual line of the left eye; a, point on the nasal side of the retina of the left eye where the image of the object is received and from which it is projected in such a way that it appears at o_2 at the left side of the true object. As shown in this diagram, the left image, o_2, belongs to the left eye, and the right image, o, to the right eye; the diplopia is therefore homonymous. If the left eye diverges, the right image belongs to the left eye, and the left to the right, the diplopia being heteronymous. (Hotz.)

Infrequency of Affections of Single Ocular Nerves.—Affections of single ocular nerves are neither common nor very rare. The sixth and third nerves are more frequently affected than the fourth by diseases limited to their trunks or nuclei. When isolated affections of the third, fourth, or sixth nerve are peripheral (trunkal) they are probably in most cases due to forms of neuritis or perineuritis, as gross lesions at the base are not likely to single out a separate nerve trunk; but it is possible that a small lesion, as, for instance, a nodosity in one form of periarteritis, an aneurism, or a small exudate, may so affect the trunk of a single nerve, or even one of the branches of such trunk at its beginning, as to produce an isolated paralysis. When not due to neuritis they are most frequently nuclear in origin. When paralysis of the third, fourth, or sixth nerve is due to a gross lesion at the base of the brain, a more or less extensive syndrome is present. Commonly several cranial nerves are conjointly affected, and, according to the location of the lesion, motor, sensory, and other tracts may also be involved, giving associated symptoms which are not referable solely to the cranial nerves. Cases showing this association of peripheral disease of the third nerve with disease of other cranial nerves are discussed in several places in succeeding pages (see pages 816, 817, and Fig. 388; also page 883 and Fig. 411).

FIG. 385.

Oculomotor paralysis, showing ptosis. The case proved to be one of gummatous meningitis, endarteritis, and thrombosis of the internal carotid; the right third nerve was bound down by exudate.

Symptomatology of Paralyses of the Third Nerve.—*Total Third Nerve Paralysis.* The appearances presented in complete paralysis of one third nerve are shown in the illustrations Figs. 385 and 386. The upper lid hangs loosely down, covering or almost covering the eye, and in order to obtain a view of the eyeball it is necessary to separate the lids by means of the fingers, as shown in Fig. 386. The upper lid can sometimes be partially raised by the overaction of the occipitofrontalis muscle. On opening the eyelids with the fingers the eye is seen to be turned outward and somewhat downward, because of the unopposed action of the two muscles which are not paralyzed, namely, the external straight and the superior oblique. The pupil is dilated and immovable, responding neither to light nor to efforts at accommodation. Usually the eye projects

slightly, because of the paralysis of three of the muscles which ordinarily hold it firmly in place. If the paralysis is confined to the third nerve, the movements of the muscles supplied by the fourth and sixth are not affected, and examination will show no loss of sensation.

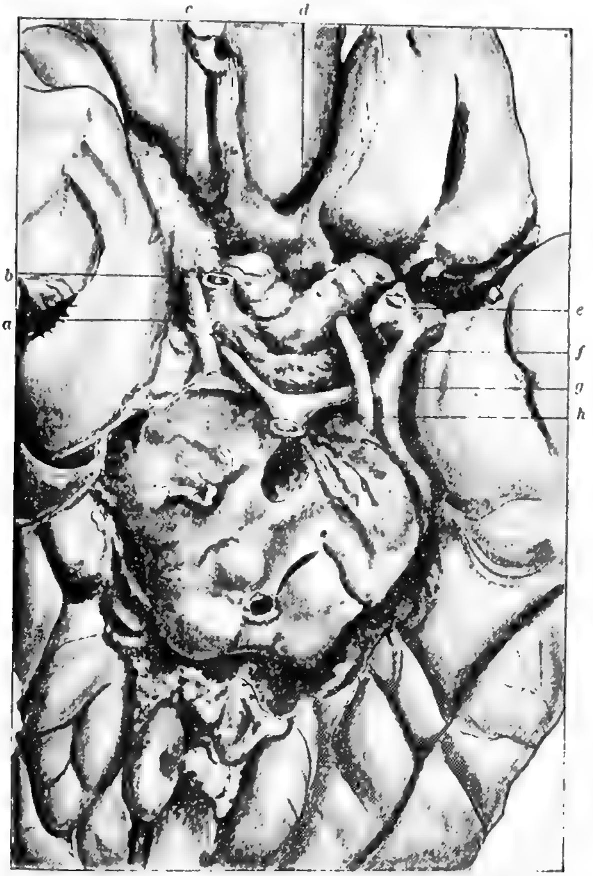
FIG. 386.

Oculomotor paralysis: the eye held open. Same patient shown in Fig. 385.

Paralysis of the Third Nerve in Cases of Multiple Syphilitic Lesions. In syphilitic, as well as in tubercular, cases, paralysis of the third nerve is not infrequently one of a series or one of a collection of more or less unrelated symptoms. The illustration Fig. 387 shows some of the gross and microscopic appearances found in a case in which the lesions were in several portions of the encephalon. Important vessels were closed by thrombi; an old gummatous mass was found in the Sylvian fossa; both optic and oculomotor nerves were compressed and atrophied; and the heads of the lenticula and caudatum on each side were softened. The symptoms were complete right oculomotor paralysis, with paresis of the left leg, and mental disturbance. The patient, who first came under observation several months before her death, was about twenty-nine years old, and had a previous history of syphilis and general dissipation. The ophthalmoplegia was both external and internal. The specimens from this case were afterwards submitted to microscopical examination, and a report, with illustrations, was made upon them by Dr. Mary Alice Schively, of the Neurological Laboratory of the Philadelphia Poly-

clinic. In the case of which an illustration is given in Fig. 388 the patient showed not only right oculomotor paralysis, but also facial paresis and evidences of meningocortical disease.

FIG. 387.



Drawing showing the lesions present in the case illustrated in Figs. 385 and 386: *a*, right third nerve adherent to fibroid mass; *b*, right internal carotid plugged by a thrombus; *c*, fibroid mass filling the Sylvian fossa and extending backward to the crus; *d*, chiasm; *e*, left internal carotid containing recently organized thrombus; *f*, left third nerve; *g*, left postcommunicant at junction with postcerebral; *h*, basilar giving off postcerebral.

Peripheral Paralysis of all the Extraocular Muscles supplied by the Third Nerve and limited to them. Cases have been recorded which show that it is possible to have paralysis of all the extraocular muscles without paralysis of either the iris or the ciliary muscle. These cases can probably be best explained by lesions situated at the beginning of the peripheral course of the third nerve, just before all its fibres have converged into one trunk, or by a lesion attacking the fibres of the trunk after they have diverged to go to their various

destinations in the orbit. Where the nerves make their exit from the brain stem the ciliary and pupillary fibres are nearer the middle line than are the others, and therefore they may remain intact when all the other fibres of the third nerve are destroyed. In these cases the symptoms are ptosis and divergent strabismus, the eye turning outward and downward, as in cases of total third nerve paralysis, but the pupils respond both to light and to accommodation.

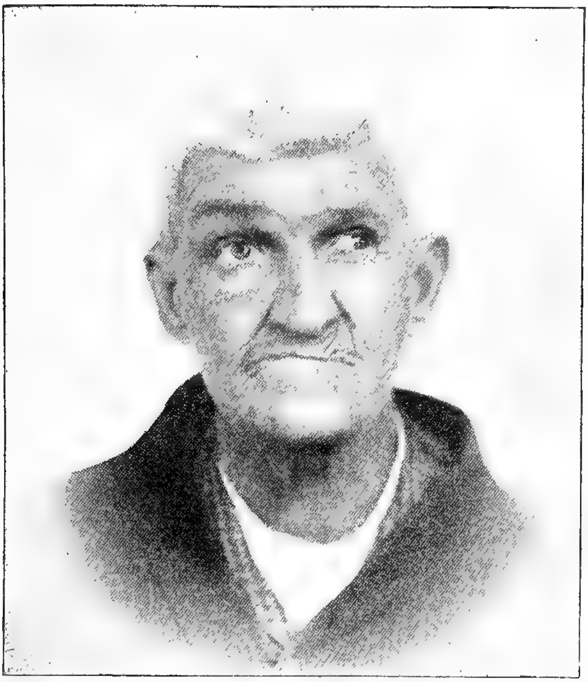
FIG. 388.

Right oculomotor paralysis, facial paresis, and meningocortical disease.

Paralysis of Single Extraocular Muscles supplied by the Third Nerve. Any one of the extraocular muscles supplied by the third nerve—the rectus internus, the rectus superior, the rectus inferior, the obliquus inferior, or the levator palpebræ—may be paralyzed alone. Paralysis of the obliquus inferior and of the levator palpebræ is uncommon, and when present the former is usually due to nuclear disease and the latter to an affection of the cortex or subcortex. Paralysis of the internal straight muscle is one of the most frequent of the single ocular palsies. When marked, the facial axes clearly diverge. Pronounced secondary deviation of the sound eye occurs when the paralyzed eye is made to fix. In paralysis of the rectus internus the head is usually turned towards the unaffected side. When the superior straight muscle is paralyzed the patient complains of double vision in looking upward, and the tendency is to throw the head backward. Single vision is present in the lower part of the field. The double images are crossed. On covering the sound eye and attempting fixation with the affected eye on an object slightly above the horizontal, secondary deviation upward of the sound eye results. Paralysis of one inferior straight muscle causes the patient to hold his head downward and somewhat inclined towards the paralyzed side. The double images are found everywhere in the lower fields, and are crossed. Paralysis of the inferior oblique is exceedingly rare, and indeed its existence as a separate affection is doubted by some of the best ophthalmological authorities, but Mauthner has reported a case which followed an injury to the muscle during an orbital operation. Theoretically homonymous double images should be caused in the upper field. The reader is referred to the paragraph on forms of ocular paralysis as studied by the diplopia tests

(page 813) for a clearer understanding of the positions of the double images in each of the different forms of paralysis of single muscles supplied by branches of the third nerve.

FIG. 389.



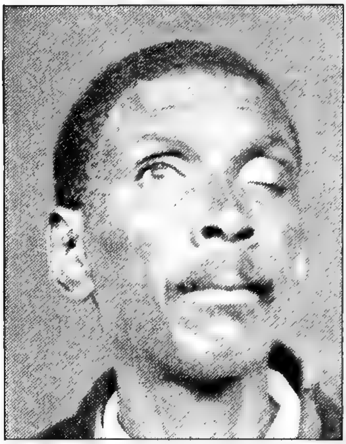
Nearly isolated paralysis of left internal rectus (left external strabismus without ptosis); both pupils responsive to light; ocular paralysis came on after scarlet fever, when the patient was one year old. (Photograph taken at the age of sixty-seven.)

Paralysis of the Levator Palpebræ Superioris. Isolated paralysis of the levator palpebræ superioris, with its symptom, ptosis, is exceedingly rare as a peripheral trunkal affection, although more common as the result of a nuclear or of a cortical or subcortical lesion. While rare as a persistent symptom of peripheral third nerve disease, isolated ptosis is often the earliest symptom of a later more complete oculomotor paralysis; it is not infrequently the first sign of such paralysis. The patient has first a slight drooping of one lid, and in the course of hours, days, or weeks the other muscles supplied by the third nerve are successively involved. The explanation of such a case as this would probably be found in the fact of the greater exposure of the peripheral distribution of the levator branch of the third nerve, or perhaps, as suggested by Jeffries, owing to its constant use its defects are more promptly recognized. It should be borne in mind that the lid is in part raised by unstriped fibres supplied from

the gangliated system, and that slight drooping of the lid occurs sometimes because of involvement of these fibres.

Bilateral Paralysis of the Third Nerve. Bilateral paralysis of the third nerve in the adult is usually of syphilitic origin. Such a case, of incomplete bilateral type, is shown in Fig. 390. This patient had complete ptosis of the left eye and partial ptosis of the right eye; movements of the eye internally, upward, and downward were completely lost, both eyes being drawn at first into the external canthi and being immovable in any direction. The pupils did not respond to light or to accommodation. The patient complained of a sensation of weakness in his lower extremities, and had an imperfect girdle sensation around the waist, but otherwise no paralytic symptoms except those of the oculomotor muscles. The third nerve paralysis in this case was most probably due to syphilitic neuritis, as a basilar meningitis or gumma would be likely to involve other structures than the third nerve. When a lesion causing bilateral third nerve paralysis is focal and superficial it is of course situated at about the point of junction of the crura, whence both third nerves proceed in their course to the cavernous sinus. In other cases, however, the lesions are deeply situated, and these are usually either focal encephalitis or plugging or rupture of small vessels. Such cases are of course rarely pure in type, as even comparatively minute lesions must involve other structures with definite functions, but the third nerve symptoms so predominate as to overshadow the others. Occasionally in syringomyelia or in progressive muscular atrophy paralysis of both third nerves is present. A few cases of congenital bilateral paralysis of the third nerves, as of other forms of unilateral and bilateral ocular paralysis, have been recorded. These are doubtless due to arrested or imperfect development of the ocular nidi. The illustrations (Figs. 391 and 392) are of a case of bilateral nerve paralysis in a child recorded by Davidson. The patient was twelve years old, and when she first came under observation had ptosis on both sides, but most marked on the right, the occipito-frontalis, as shown in Fig. 391, contracting in order to counteract the lack of power in the elevators of the eyelids. Divergent strabismus was present in both eyes, most marked on the right. All the external muscles of the eyeball supplied by the third nerves of both eyes were more or less paralyzed. The pupils were active and re-

FIG. 390.



Bilateral oculomotor paralysis.

sponded normally, and accommodation was unaffected. Vision and ophthalmoscopic appearances were also normal. Later in the history of the case the pupils became moderately dilated and did not react to light; still later the response to light returned in both eyes, but

FIG. 391.

Bilateral paralysis of the extraocular muscles supplied by the third nerve. (Davidson.)

FIG. 392.

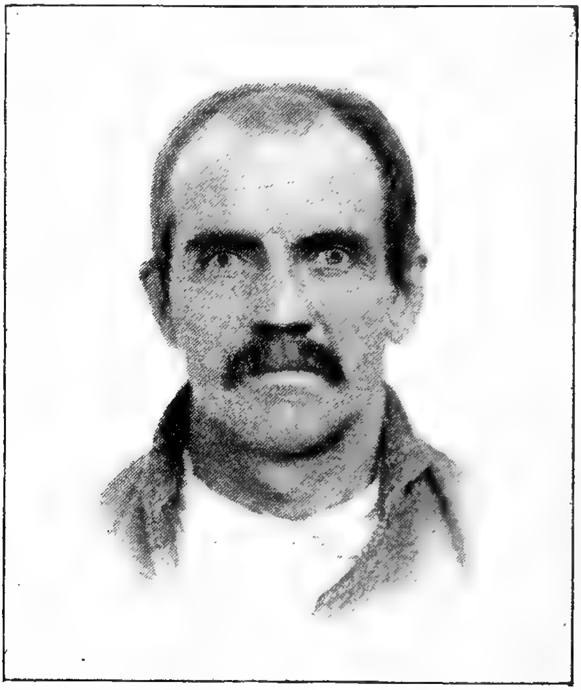
Photograph of case shown in Fig. 391, taken five months after recovery from bilateral paralysis of the extraocular muscles supplied by the third nerve. (Davidson.)

both failed to react to accommodation. Under the use of potassium iodide, after one relapse, she was discharged cured. In Fig. 392 this child is shown as she appeared five months after she was finally discharged.

Affections of the Intraocular Muscles supplied by the Third Nerve.—*Effects of Focal Lesions variously situated in the Optic Pupillary Pathway.* The special effects of lesions of the optic pupillary pathway vary according as these lesions are destructive or irritative, unilateral or bilateral, and according as they are situated in the centripetal, central, or centrifugal portion of the reflex arc. They can be best understood by a careful study of "the scheme of the nuclei of the nerves of ocular movements, and of their central and peripheral parts" (Fig. 383, page 808). When the nuclei of both the sphincter iridis and the ciliary muscle are the seats of destructive lesions, paralysis of all the intraocular muscles results. When one nucleus alone is affected, the muscle supplied by the nerve which originates in this nucleus is paralyzed, causing either dilatation and rigidity of the pupil or loss of accommodation. Spasmodic affections of the intraocular muscles result from irritative lesions of the nuclei of the nerves to these muscles, but they are rare. When the intraocular

portion of the optic oculomotor tract is diseased in the pregeminum or in the tracts which connect the pregeminum with the nucleus of the sphincter of the iris, the pupils do not react to light, but the reactions to accommodation and convergence remain. True disturbances of vision are absent. When the connections of the pregeminums with the sphincter nuclei are diseased, the reflex rigidity of the pupil (loss of reaction to light) with normal reaction to accommodation and convergence is present. Some interesting cases illustrating varieties of *consensual iritic reaction* have been observed, and

FIG. 393.



Right iridoplegia (dilatation of right pupil); paresis of convergence: accommodation normal: interference with consentaneous action of pupils—direct light stimulation of the right eye produces no pupillary reaction in this eye, but reflex response in the left eye; light thrown into the left eye produces pupillary reaction in this eye, but no response in the right eye. In the diagram Fig. 394 the probable position of the lesion causing the iridoplegia is shown at *A*.

some results of general clinical application have been obtained especially by Redlich in the course of his studies of paralytic dementia. He found that in all cases when each iris responded to direct light stimulation the consensual reaction was intact in each eye. If, for instance, the right and left pupil each responded to direct light stimulation, light thrown into the left eye caused response in the right, and light thrown into the right eye caused response in the left. If neither iris responded to the stimulation of light, consensual re-

action was absent in both irides. In all cases in which the one iris failed to respond to direct illumination the consensual reaction in the other eye could be elicited; but in these cases when the sound eye was exposed to light no pupillary response was induced in the iridoplegic eye. Redlich's conclusion from his observations on these cases was that a partial decussation of the optic iritic fibres takes place, so that from each optic nerve fibres proceed both to the right and to the left oculomotor nuclei. In the cases cited by him the lesions must have been situated centrad of the decussation, which probably takes place in the chiasm or postcommissure. These peculiarities of consensual iritic reaction are seen in other diseases than paretic dementia, and may indeed occur as the results of any disease giving a lesion of the optic iritic tract or its centres. An interesting case of this kind was a patient at the Philadelphia Hospital, a photograph of whom is shown in Fig. 393. Examination of this case showed no hemianopsia, but general contraction of the visual field.

The Position of Lesions in the Optic Pupillary Pathway with Reference to the Production of the Hemiopic Pupillary Reaction.—Wernicke's hemiopic pupillary reaction (or inaction) has been described and the method of determining it explained on pages 160 and 161, and references to this pupillary phenomenon have been made in other places. It will only be necessary to refer here to the positions in the pupillary arc of lesions which would or would not cause this interesting diagnostic sign. The following is a summary of Henschen's conclusions regarding this matter. The reaction is caused (1) by mere pressure on the optic tract; (2) usually by lesions of the optic tract, even when they are very minute; (3) by lesions of the chiasm, although occasionally for some unknown reason it is not present with such lesions; (4) by injury to the optic nerve with monocular hemianopsia; and (5) by lesions of the posterior segment of the thalamus and pulvinar, it being here probably the result of pressure on the optic tract or of destruction of the brachium anterius (prebrachium). The sign is not produced (1) with destructive lesions, like softening of the occipital, parietal, or temporal lobe, even when these are extensive; and it is absent even in tumors of these regions which destroy the optic

FIG. 394.

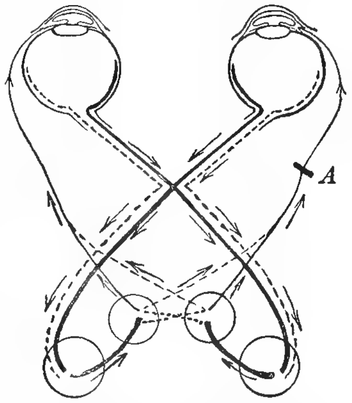


Diagram showing the probable position of a lesion (at *A*) in the course of the optic iritic tracts in a case of right iridoplegia, in which the iris on the paralyzed side failed to respond to direct light stimulation, the left pupil responding both to direct and to indirect stimulation. The larger circles represent the quadrigeminal bodies, and the smaller the centres for the iritic sphincters; the arrows indicate the directions of the afferent and efferent impulses.

radiations and cause considerable pressure; (2) by lesions of the postgeniculum; (3) by destruction of the pulvinar; (4) by destruction of the postgeminum. The effect of lesion of the pregeniculum is, according to Henschen, uncertain.

The Cutaneous Pupillary Reflex. Strong sensory stimuli applied to the skin almost anywhere may produce dilatation of the pupil, but this effect is particularly brought about when the skin of the neck is stimulated, as, for instance, by an electrical current. The path for the completion of this reflex is supposed to be by way of the cervical gangliated system, the ciliospinal centre in the cord, and the pathway through the oblongata which connects this region with the centre for the sphincter iridis. Lesions of any portion of this arc may of course produce either dilatation or contraction of the pupil according to their character.

Pupillary Phenomena resulting from Lesions involving the Fifth Nerve. Both dilatation and contraction of the pupil have been noted in connection with lesions of the fifth nerve trunk and also of its roots, especially of its great spinal root. It has been suggested that in some cases at least these pupillary disturbances are of a reflex character, that is, are the result of irritation reflected to the pupillary centres and fibres. Whether this explanation is or is not correct for some of the cases, it is probable that in other instances the pupillary change is produced by lesions which, while involving the trigeminal roots, implicate the tract in the oblongata associating the spinal and the encephalic pupillary regions. When trigeminal anesthesia is associated with reflex pupillary immobility, Turner holds that it is due to a lesion of the spinal (formerly called the ascending) root of the fifth.

Haab's Cortical Pupillary Reflex. It was found by Haab that contraction of both pupils took place when attention was suddenly directed to a bright object in a somewhat darkened room. The amount of contraction is proportionate to the brightness of the object. The pupillary contraction is independent of any distinct change due to convergence or accommodation. Owing to the fact that the contraction follows a purely psychical process, Haab believed that it was in all probability of cortical origin. The occurrence of this reflex presupposes that the network of the motor corona radiata to the motor oculi nucleus, and the ganglion cells of the nucleus of the sphincter, are intact. "The cells or fibres of the accommodation nucleus may be incapable of function, and Haab reports two cases in which the reflex was retained despite paralysis of accommodation. The reflex will be lost if the internuclear fibres between the nuclei of the ciliary muscle and pupillary sphincter are destroyed, even though both nuclei are intact. In such a case accommodation and the involuntary light reflex of the pupil may be normal, and this has also been observed by Haab." (Knies.)

Ciliary Spasm. Ciliary spasm is of frequent occurrence in connection with disorders of refraction, especially with hypermetropia or hypermetropic astigmatism. Efforts to accommodate for near vision in these instances sometimes produce a strong and persisting cramp, which may be so great as to mask the real degree of optical defect which is present. Under mydriatics this spasm disappears, and the latent disorder of refraction comes to the surface. Ciliary spasm is also sometimes due to the instillation of drugs like eserine or pilocarpine, and occasionally this form of spasm may be of severe type. Usually it passes off promptly after discontinuance of the instillation. Valk has reported true tonic spasm of accommodation occurring in a case of marked myopic astigmatism. The eyes were sensitive to light. The use of atropine four grains to the ounce almost continuously for five months gave marked relief. During the treatment the patient was given strychnine in small doses for a month, and twice the temples were leeched. Patients suffering from ciliary spasm have a feeling of discomfort in the eyes, not often of the nature of real pain, but sometimes described as one of drawing or contraction. The light, frequently complained of, probably acts as an irritant and an augmenting factor in the induction of the spasm. In rare cases acute ciliary spasm is unconnected with disorders of refraction or with the use of drugs, and such cases may be due to irritative lesions of the accommodation centre. In some forms of disease of the spinal cord, as myelitis, sclerosis, or tumor, a severe ciliary spasm with myosis is present, and is probably due to irritation reflected to the accommodation centre. Spasm of accommodation may be due to cortical irritation. The centre of representation of macular or acute vision, while situated in the general visual sphere, is separated from that for the representation of associated halves of the retina. Obregia noted that irritation of the macular region of the visual sphere excited scarcely any movement, but that the conjugate movements of the eyes towards the opposite side became more extensive the nearer the periphery of the visual sphere was approached. If the image of an object falls upon the fovea centralis of the retina and is conducted thence to the macular portion of the visual sphere, no conjugate movement will result, because the eye is already adjusted; convergence and accommodation are alone necessary, and for these two movements the macular portion of the visual sphere must be regarded as the cortical centre. If a separate cortical centre for macular representation exists, and if this centre is also the centre or contains the centre or centres concerned with convergence and accommodation, it follows that a focal irritative lesion of this region might produce ciliary spasm with contraction of the pupil, narrowing of the eyelids, and reduction of acuity of vision. While, so far as I know, such cases have not been recorded, it is easy to see that they might have been overlooked. If a focal cortical lesion results in

ciliary spasm, it follows that a lesion of the fibres associating the macular and the oculomotor cortex, or of the projection fibres from the cortex to the ciliary or accommodation nucleus, might also cause ciliary spasm.

Paralysis of the Associated Motion of the Eyes in Convergence. It has already been stated that a centre of convergence (Perlia's central nucleus) is located far forward in the oculomotor nucleus, between the centres for the internal straight muscle and for accommodation. Paralysis of near vision, or conjugate convergence paralysis, is, as suggested by Jeffries, the obverse of lateral conjugate paralysis. Neither eye will turn in when both eyes are opened, but if one is covered the other turns in at once, while the other turns out to a parallel position. Benzler records the case of a bandman who while playing on the horn suddenly lost sight of his music. Careful examination showed that distant vision was good, but near vision was poor and unaccompanied by crossed diplopia. When one eye was covered, the other turned in well for near vision, the covered eye turning out at the same time. The right pupil reacted to light, but not to accommodation. (Jeffries.)

Spasm in Convergence. Millingen has reported a case (cited by Spitzka) in which a focus of disease situated to the right of the aqueduct pressed close on the median line of the central tubular cinerea. During life the patient had suffered from spastic (irritative) contracture of both internal straight muscles. To account for this case it is necessary to assume an irritative lesion of the cell nests related to both internal straight muscles, or, what is more probable, of Perlia's central nucleus (the convergent centre).

Affections of Ocular Movements due to Disease limited to the Fourth Nerve or its Nucleus.—*Symptoms of Fourth Nerve Paralysis.* Disease limited to the trunk of the fourth nerve or its nucleus is comparatively rare. When the disease is unilateral a form of strabismus is produced in which the eye is rotated slightly upward and inward. Double images appear when the eyes are turned downward. The diplopia is homonymous, and the images are placed one above the other. The image on the paralyzed side is lower than that on the other, and is inclined towards the unaffected side. The images become more widely separated both vertically and laterally, and the test object is carried farther downward and outward. Often squint is so slight that it will be overlooked on mere inspection, but it will be clearly shown by careful testing for double images. When the paralyzed eye is fixed by covering the sound eye, secondary deviation of the sound eye downward and inward occurs. The patient inclines to turn the head forward and towards the healthy side to correct the false projection, which is downward and a little outward. This form of paralysis frequently produces marked vertigo, and causes great confusion to the patient, owing to

the curiously placed double images in the lower field. The third and fourth nerves are not infrequently paralyzed together.

Double Fourth Nerve Paralysis. As has been shown, the fourth nerve makes a complete, or nearly complete, decussation which is readily traced just above the posterior extremity of the aqueduct. Should a lesion happen to affect this place of crossing, the chiasm of the fourth nerve, the result would be a double paralysis of the superior oblique. I have not been able to discover any cases of this kind in the literature to which I have had access. Double fourth nerve paralysis may also be caused by lesions involving both nuclei or both trunks of the fourth nerve, and may be anticipated especially in some cases of multiple syphilitic lesions.

Affections of Ocular Movements due to Disease limited to the Sixth Nerve or its Nucleus.—*Unilateral Paralysis of the Sixth Nerve.* Isolated paralysis in the domain of the sixth cranial nerve vies in frequency with cases of third nerve paralysis. When the paralysis is unilateral it gives rise to convergent strabismus on one side; when bilateral, to double convergent strabismus. In the unilateral cases double images are likewise seen when the eyes are turned in the horizontal plane towards the paralyzed side. If the paragraph which describes the diplopia tests be consulted, the position of the different images in cases of this form of paralysis will be clearly seen. The distance between these images increases according as the object is moved towards the paralyzed side. Like isolated paralysis of the third and fourth nerves, it is most frequently due either to a peripheral neuritis or to nuclear lesion. It is a comparatively common accompaniment of the other symptoms of the early stages of tabes, and transient forms of abducens paralysis of unknown origin are observed especially after sixty years of age. Occasionally it results from traumatisms directly affecting the muscle or nerve. In marked cases of abducens paralysis

Fig. 395.

Paralysis of the left external rectus and paresis of the right external rectus; accommodation good; vision reduced to about one half in the right eye, to about one third in the left; abducens root fibres implicated in the lesions as found on autopsy and microscopical examination.

the patient cannot turn the eye beyond the median line, and vertigo, with or without nausea and vomiting, is often severe.

Bilateral Paralysis of the Sixth Nerve. Bilateral paralysis of the muscles supplied by the sixth nerve is rare in any form, and is especially infrequent as an acquired disease in the adult. It may, however, occur as the result of a nuclear ophthalmoplegia limited to both nidi of the sixth pair, or to multiple lesions which by unusual chance affect only the tracts for the peripheral distribution of both sixth nerves. As a congenital disorder it has been observed in a number of recorded instances. Leszynsky, in recording a case, has also given a summary of the literature of this subject. His patient was four years of age when first studied. Towards the end of his first year he began to draw his head backward, and his mother then noticed that his eyes both turned inward. Examination showed that the convergent strabismus present in both eyes was more pronounced in the left. He was unable to move either eye outward beyond the middle line. About a dozen cases corresponding more or less closely to that of Leszynsky have been recorded. One case associated with bilateral facial paralysis, reported by Harlan, was also seen and studied by the writer.

General Remarks on the Focal Diagnosis of Disturbances of Ocular Movements.—As the subject of cerebral localization has already been considered at length, our concern here will be more with the peripheral and nuclear affections, and yet it will not be possible to understand clearly diseases of the oculomotor apparatus unless they are all glanced at in the same connection. Certain principles of comparatively easy application can be used in differentiating affections at different levels in the oculomotor system, which includes the whole apparatus related to ocular movements from the surface of the brain to the motorial end plates in the intraocular and extraocular muscles. With one exception—ptosis from paralysis of the levator palpebræ—lesions of the cerebral cortex and subcortex never cause paralytic or spasmodic affections of single ocular muscles. When these are present, therefore, lesions of the nuclei, root fibres, or nerve trunks at the base should be expected. As the coordinations of ocular movements are in a certain degree determined by the action of tracts which associate cortical centres together or bind these centres with basal regions, lesions of the subcortex or internal capsule, crus, or pons may give rise to disturbances of these coordinations, and these may be either of a paralytic or of a spasmodic type. Other points of particular diagnostic value will appear as focal lesions at different sites are considered. Consulting the scheme Fig. 383, page 808, it will be seen that the ptosis in the cortical cases, to which reference has just been made, will be caused by lesion of P, the cortical centre for the levator palpebræ superioris. As to what would be the effect of the destruction of the cortical oculomotor centre of

one hemisphere, few clinical facts are at command. Destruction of one oculomotor centre should cause total paralysis of all muscles supplied by the nerves whose nuclei are in connection with the destroyed cortical area. These nuclei include all those which are situated on the diseased side, with the exception of the nucleus of the levator palpebræ and the nuclei of the fourth and sixth nerves. The cerebrobulbar ocular tracts decussate before passing to the fourth and sixth nuclei. So also does probably the nerve branch to the nucleus of the inferior oblique muscle. If, therefore, the right oculomotor centre is destroyed, all the ocular movements of the right side, with the exception of those performed by the inferior oblique and the levator palpebræ, are paralyzed, but the movements produced by the right abducens and the right trochlearis are not paralyzed. On the left side the movements performed by the abducens or trochlearis and the inferior oblique are affected, while the others are performed normally. The clinical result will be that conjugate movement of the eyes towards the left will be impossible, while convergence of both eyes can be performed. This convergence is possible because the convergent nucleus (Perlia's central nucleus) is innervated from both oculomotor cortical centres. The transient conjugate deviation in cerebral apoplexies is probably due to destruction of the cortical oculomotor centres or of the subcortical oculomotor tracts. Irritation of one oculomotor centre or tract causes deviation of both eyes towards the opposite side. In some cases of diplopia the effects of both irritation and destruction are commingled and cause a confusing picture.*

Disorders of Ocular Movements due to Cortical Lesions.—Herter has reported a case in which unilateral ptosis was apparently due to a limited cortical lesion of the opposite hemisphere. The right arm and right leg were flaccid, the facial muscles not being affected. The limbs of the left side showed no weakness, but ptosis, not quite complete, was present on this side, and the left pupil was slightly dilated, reacting poorly to light as compared with the right. The autopsy showed extensive pulmonary tuberculosis and suppurative nephritis. In the right hemisphere, just below the intraparietal fissure was a circular patch of softening, one inch in diameter, occupying the angular convolution. The softening involved the cortex, and to a slight extent the substance beneath it. Herter believed that it was safe to refer the left-sided ptosis to this lesion in the right angular convolution, and that the right-sided hemiparesis may have been due to uræmia associated with the kidney disease.

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* Some of the diagnostic points given here, and also when the lesions of the optic pupillary pathway were considered, have been taken from Magnus (Anleitung zur Diagnostik der centralen Störungen des optischen Apparates, Breslau, 1892).

Cases of one-sided ptosis due to cortical lesion have been reported by other observers. About thirty cases of ptosis, either congenital or acquired, probably due to corticular arrest or lesion, have been placed on record (Swanzy). The left lid is most frequently affected. In most of the cases referred to by Swanzy the ptosis has been combined with associated movements of the affected eyelid. At least three conditions have been observed, namely, elevation of the drooping lid when the eye is abducted, when the eye is adducted, and when the mouth is open. In some cases a synchronous contraction of the pupil has been noticed, while in others a lateral motion of the jaw and movements of deglutition are accompanied by elevation of the lid. According to Swanzy, ptosis has no value as indicating the locality of a lesion in the cortex, but when monolateral and the only focal symptom it occurs with cortical lesions alone. In not a few cases it is probably a distant symptom.

Disorders of Ocular Movements due to Limited Subcortical (Cerebral) Lesions.—Very few cases have been put on record in which limited lesions of the corona radiata or of the internal capsule have caused disorders of ocular movements. The symptoms of destruction of the oculomotor tracts, as stated in the last paragraph, are probably similar to those of one of the oculomotor centres. It is probable, however, that these symptoms will be of a more persistent character than when the lesions are purely cortical or nearly so. If, for example, the oculomotor tract for the right side is destroyed, conjugate movement of the eyes towards the left will become impossible, although the power of convergence may remain. If the lesion of these tracts is more irritative than destructive, the conjugate deviation of the eyes will take place towards the opposite side, and I believe that this will be the case even when the lesion is above the pons. It will be necessary to discuss briefly some of the points connected with cerebral coordination of ocular movements, and to give in detail some of the facts regarding affections of consentaneous ocular movements.

Ptosis from Lesions of the Striatum.—According to Nothnagel, ptosis or pseudoptosis with a peculiar train of accompanying symptoms has been found in lesions of the striatum. In these cases the symptoms are (1) apparent ptosis of the paralyzed side, due to contraction of the palpebral aperture; (2) contraction of the pupil of the same side; (3) an apparent shrinkage back of the eyeball into the orbit; (4) an abnormal secretion of mucus from the corresponding nostril, of tears from the eye, and of saliva from the corresponding side of the mouth. (Swanzy.)

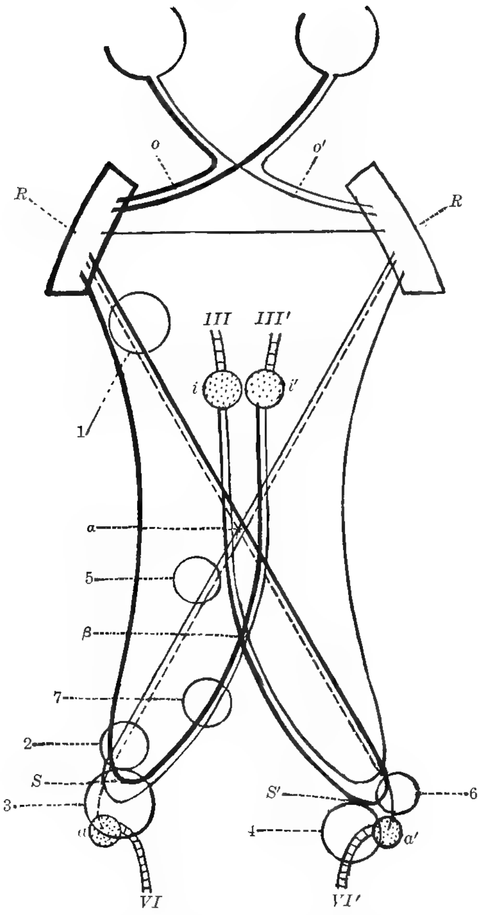
Cerebral Coordinations of Ocular Movements.—The chief cerebral coordinations of ocular movements are four in number: (1) movements of both eyes to the right; (2) movements of both eyes to the left; (3) movements of both eyes downward and inward,

narrowing of the pupils, and contraction of the ciliary muscles, producing increased convergence and accommodation; (4) movements of both eyes upward and outward, producing diminished convergence, and accompanied by, though not actively producing, a widening of the pupils and relaxation of accommodation. These several forms of compound movements are produced by the action of distinct brain centres (Priestley Smith). Theoretically, any one of these forms of coordinating movements may be affected by either paralysis or spasm. Affections of lateral movements are by far the most frequent. As a rule, eyes and head are moved together, but the former may alone be affected.

Some of the Varieties of Conjugate Deviation and their Mechanism.—*Conjugate Deviation due to Cortical and Subcortical Lesions.* Numerous physiological observations have shown that conjugate deviation (sometimes called Prevost's symptom) may result from irritation of almost all parts of the cerebral cortex; but exact experiments have determined that such deviation can be obtained with most certainty from irritation of the occipital lobe,—in other words, from irritation of the visual sphere. In a few cases this deviation remains constant after a cerebral hemorrhage, or is a persistent symptom the result of some other form of focal lesion. Wernicke held that such deviation coming on with shock and without loss of consciousness pointed to disease of the lower temporal region of the opposite side, but Jeffries regarded Wernicke's cases as unsatisfactory. In a very small number of cases lesions of the frontal convolutions have been accompanied by disorders of ocular movements of such a kind as to suggest some connection with the lesion. The supposed positions in the internal capsule of the tracts containing the motor fibres for these movements are seen in Fig. 236, page 360, where they are placed as if located just in front of the knee of the capsule, and are designated by the expressions "eyes opened" and "eyes turned." The fasciculus conveying fibres concerned with the movements of the turning of the head is represented as just at the knee of the capsule.

Conjugate Deviation due to Pontile Lesions. Bleuler, in 1885, collected a series of cases of conjugate deviation due to pontile disease. Jeffries, in 1892, nearly doubled the number of such cases. He summarized as follows the different ocular states found as the result of such lesions: (1) complete paralysis of one external rectus and a crossed paralysis of the internal rectus, so that neither muscle is capable of motion, the eyes looking forward or to the other side according to the state of the opposite pair of muscles, in these cases the eyes turning readily to the opposite side, up or down, but stopping at the midline as if transfixed; (2) precisely the same state as in the first class, except that the internal rectus acts perfectly for near or for convergent vision; (3) when both eyes are open, the eye with

FIG. 396.



Bleuler's diagram in explanation of the mechanism of conjugate paralysis: *o*, *o'*, optic tracts; *R*, *R'*, cortical centres; *a*, *a'*, abducens nuclei; *i*, *i'*, internal rectus nuclei; *S*, *S'*, point where the internal rectus fibres bend; 1, lesion producing Prevost's symptom; 2, 3, lesions producing paralysis of one abducens and the opposite internal rectus for all movements; 4, lesion producing paralysis of one abducens and weakness of the opposite internal rectus for all movements; 5, paralysis of left abducens, absolute defect of right internal rectus for associated lateral movements, weakness of both internal recti for convergence; 6, paralysis of abducens and inactivity of opposite internal rectus for lateral movements only; 7, total paralysis of opposite internal rectus; α, double abducens paralysis; β, paralysis of convergence; according to the view of Bleuler, fibres run direct from the cortex to their respective root nuclei, but there are two sets of fibres for the internal rectus, one for conjugate vision and one for near vision; the fibres run down as separate bundles below their nucleus close to the sixth nucleus, and then turn back and cross to their nucleus. (Jeffries.)

the paralyzed internal rectus does not turn in for objects on the other side of the nose, but does if the other eye is covered; (4) the internal rectus does not act in conjugate but does in near vision, the external rectus being normal; (5) though devoid of any signs of conjugate paralysis, simple nuclear sixth nerve paralysis must be considered as an element of conjugate paralysis. Jeffries found it especially difficult to explain those cases in which when both eyes are opened the eye with the paralyzed internal rectus does not turn in, but turns in when the other eye is covered. He believed that the only way out of the difficulty was to assume that the apparent conjugate movement in monocular vision is not in fact this, but really a substituted convergent movement.

Conjugate Deviation due to Tumor (Irritative Lesion) of the Pons. The most striking illustrations of spasmodic conjugate deviation due to encephalic lesion have been afforded by disease of the pons, and especially tumors in this location. In 1882 I reported an interesting case of this kind, in which the chief

symptoms of focal lesion were hemiparesis, partial right ptosis, diminished sensation on the left side of the face and in the right limbs, conjugate deviation of the eyes and rotation of the head to the right, persistent epistaxis, and a tendency to hemorrhage from the mucous membranes. The autopsy revealed a gumma about half an inch in diameter, distinctly limited to the left cephalic quarter of the pons. Other similar cases have been recorded. When the lesion is irritative, the deviation is towards the side of the lesion; when it is destructive, it is away from the side of the lesion.

Affections of Upward and Downward Rotation of Both Eyeballs. Cases in which the movements of both eyes upward or downward are affected are comparatively rare, and they are usually paralytic in type. Jeffries refers to five of these cases, and at least as many more have been reported under various names, such as paralysis of the superior recti muscles, paralysis of upward rotation, paralysis of the inferior recti, and paralysis of downward rotation or downward movement. In nearly all cases with autopsies lesions have been found at about the level of the third pair, but Thomsen has reported a case in which the conjugate paralysis of movement was apparently due to a gumma at the point of exit of the third nerve between the albicantia and the crura. The nuclei of the third nerve were normal. Bruner, under the title of "paralysis of the superior recti muscles," has reported a case in which intermittency in the relative strengths of the two paretic muscles was shown. Nothing in the case indicated spasm of the inferior oblique, and two degrees of esophoria were present. The reporter of this case inclines to the view that the paralysis was congenital, and believes that the condition was due to a central deficiency rather than to malformation or arrested development of the muscles. Recently in consultation with Dr. Elwood Patrick, of West Chester, and Dr. H. F. Hansell, of Philadelphia, I saw an interesting case of paralysis of the movements of upward rotation of both eyes. In this case the trouble with vision began about four years before coming under observation, and for nearly half this time the patient had been troubled with diplopia. According to the report received from Dr. Hansell, since two years before, when first seen by him, the patient had had paralysis of upward deviation of both corneas, it being impossible for him to raise his eyes beyond the horizontal plane. The left eye diverged slightly, but there was no true lateral paralysis. The pupils were equal, reacting to light and accommodation both individually and consensually, but their movements were sluggish. The right field was concentrically limited to a decided degree, and slight concentric limitation was present on the left. The media and fundus were healthy. The central acuity of vision was 20/40 in the right eye and 20/30 in the left. Examination showed slight difficulty in speaking. The tongue was protruded slightly and a little to the left, and the patient showed

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some tendency to drooling. He complained of a general feeling of weakness in both legs. Both knee jerks were exaggerated, and a slight, probably spurious, ankle clonus was present on the left. It is not improbable that in this case the lesion present was one of the nuclei or root fibres, and was similar to the lesions recorded in the few cases in which autopsies have been placed on record.

Nuclear Ophthalmoplegia.—*Varieties.* The subject of nuclear ophthalmoplegia is one that has claimed much attention from neurologists and neuroophthalmologists. The term is strictly correct if properly applied, but many of the cases which are classed as "nuclear"—and this remark applies to other forms of so-called nuclear paralysis as well as to the ocular palsies—are not solely nuclear, and some of them perhaps are not nuclear at all. In Wernicke's poliomyelitis superior, and in some other forms of focal lesion of the pons which are said to give rise to nuclear ophthalmoplegia, lesion destroys nerve roots as well as nerve nuclei, and indeed not infrequently involves structures other than those related to the ocular muscles. These cases are examples of nuclear ophthalmoplegia and something more. Radicular or fascicular ocular palsy, either of which may in its symptomatology very closely simulate ophthalmoplegia due to nidal lesion, is sometimes classed as nuclear. A true nuclear ophthalmoplegia is a paralysis which is due to a destructive or a degenerative lesion which expends its baneful influence on the cell nests proper and on these alone, and is best represented by chronic slowly progressive degenerative disease which gives similarly chronic and slowly progressive degenerative paralysis of the ocular muscles. In the first place, nuclear ophthalmoplegia, whether pure or mixed, may be divided into *acute* and *chronic* forms. Each of these varieties may be subdivided according to the exact locality and limitation or diffusion of the lesion producing the paralysis. Nuclear ophthalmoplegia may, for example, be *unilateral* or *bilateral*, *internal* or *external*, *isolated* when confined to one nucleus or one set of nuclei, and *total* when all the oculomotor nuclei are implicated. Objections have been made to this differentiation into special focal varieties, but they are certainly useful for practical diagnostic purposes.

Acute Nuclear Ophthalmoplegia. Acute nuclear ophthalmoplegia presents itself in several varieties, according to the nature of the attack and the extent of its ravages. In a few cases it is abrupt, and it may be fulminant, rapidly destroying all the ocular centres and the life of the patient by the invasion of other vital portions of the pons and oblongata; or it is comparatively slow in onset, and, while serious in its results, recedes after an indefinite amount of permanent damage has been done to the nuclei of the nerves of the ocular muscles, and also in some instances to adjoining parts. In a third class of cases the disease, while more or less acute in its

onset, is comparatively mild in its results, the patients making at least approximate recoveries, slight defect of motion in some of the muscles affected being the only lasting evidence of the attack. In still another class it would seem from recorded instances that the affection is purely functional, probably a form of nuclear neurasthenia. In the severe type of acute nuclear ophthalmoplegia the patient may have marked fever, and convulsions are of frequent occurrence. The acute form of ophthalmoplegia may be associated with some symptoms of spinal disease, like poliomyelitis, or with evidences of more diffuse bulbar disease, such as facial, glosso-pharyngeal, or hypoglossal paralysis, or indeed paralysis of any of the cranial nerves. Under the name of *polioencephalitis superior*, Wernicke long since described a form of paralysis in which the ocular muscles were chiefly but not exclusively involved. The cases included in his description would be classed by us under mixed nuclear paralyses,—that is, nuclear paralyses associated with conditions due to destruction of adjoining structures, such as the nerve radicles and pontile tracts. The affection is entirely analogous to the other forms of focal encephalitis attacking the bulbar region, such as those which produce the so-called polioencephalitis inferior and various irregular types of bulbar paralysis. Not infrequently ophthalmoplegia due to a polioencephalitis superior expands into a more complete form of bulbar paralysis, or other forms of bulbar paralysis late in their history include ophthalmoplegia in their syndrome.

Chronic Nuclear Ophthalmoplegia. Chronic nuclear ophthalmoplegia, like acute nuclear ophthalmoplegia, may be of several varieties, according to the mode of onset, the method of progression, and the nuclei simultaneously or successively attacked. Thus we may have (1) a chronic form in which one nucleus after another is slowly invaded until the whole nuclear oculomotor substratum is destroyed or nearly destroyed ; (2) a form in which two or more of the ocular nuclei are attacked slowly but practically at the same time ; and (3) a form in which a chronic condition of partial paralysis is left after an acute nuclear ophthalmoplegia (although perhaps in a strict sense this class should not be included under forms of chronic nuclear ophthalmoplegia). Chronic nuclear ophthalmoplegia may be associated with such diseases as multiple or combined sclerosis, Friedreich's ataxia, tabes, paretic dementia, progressive muscular atrophy, syringomyelia, and bulbar paralysis of a general type. One of the distinctions usually made in chronic nuclear ophthalmoplegia is between the *stationary* and the *progressive* form. Most of the so-called stationary chronic ophthalmoplegias do not, of course, in a strict sense deserve this name. A certain degree of ophthalmoplegia, usually symmetrical and confined to the extraocular muscles, is reached after a variable but comparatively short period ; then little or no

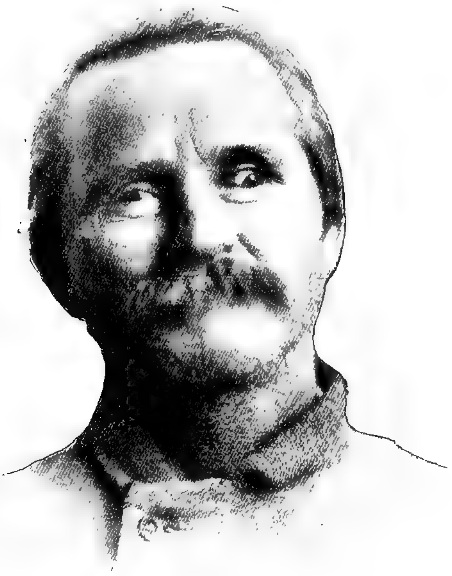
progression is made for another period, also variable, but often quite long,—it may be months or years. The symptoms have remained stationary for at least ten years. In the genuine progressive variety the disease slowly but surely saps the vitality of the whole nuclear apparatus. A form of what might be termed congenital chronic nuclear ophthalmoplegia is somewhat rarely observed. Most frequently it is bilateral and involves only a limited number of the nuclei. Double congenital ptosis is a comparatively common affection, and may be nuclear. When cortical it is much more frequently unilateral. Instead of double ptosis the congenital nuclear affection may be a bilateral paralysis of the abducens or of any one of the series of extraocular muscles, although such double congenital paralysis of the fourth nerve is extremely rare. Either the nuclear paralysis of the intraocular muscles is an affection of extreme rarity or the cases have not been observed or recorded. It is often a matter of exceeding difficulty to decide in congenital cases whether cortex, subcortex, nucleus, nerve trunks, or muscles were the parts primarily attacked. A form of chronic nuclear paralysis is occasionally seen in young children who have apparently been born with normal ocular apparatus. Such cases may occur in more than one member of the same family, and they probably simply represent another type of the so-called family affections of teratological or degenerative character.

Radicular or Fascicular Oculomotor Paralysis.—It is a rare occurrence to find an ocular or any other form of cranial nerve paralysis caused by a lesion strictly limited to the root fibres of the nerve affected. Although the course of these radicles is in some instances long,—as, for example, in the case of the abducens,—the rootlets run in such close connection with other important structures that the paralysis is not likely to be isolated. It is therefore a diagnostic point of some importance to know that when abducens or fourth nerve paralysis, but especially the former, is associated with facial or trigeminal symptoms of the same side, and anesthesia or paralysis of the opposite side referable to lesion of the lemniscus or pyramidal tract, the lesion producing this combination of phenomena, so far as the ocular affection is concerned, is probably radicular. In other words, some of the forms of alternate hemiplegia—cranial nerve paralysis of one side, with paralysis of the extremities of the other—are produced by lesions which involve the root fibres of the cranial nerves.

Alternate Hemiplegias of the Ocular Type.—The best known forms of alternate hemiplegias are those in which the arm and leg of the opposite side and the face of the same side as the lesion are paralyzed. These alternate hemiplegias have already been considered (page 572). It will only be necessary here to direct renewed attention to the fact that lesions situated on the ventral surface of the

crus at about its junction with the pons or in the cephaloventral segment of the pons may give rise to the oculomotor or third nerve type of alternate hemiplegia or to a paralysis in which both third and fourth nerves may be affected on the same side, and the face, limbs, and body on the opposite side. Alternate hemiplegia involving the abducens alone is necessarily very rare, owing to the close relations which this nerve has with the facial and auditory nerves at its nucleus, in its course, and at its superficial origin. In such a case paresis of the rectus externus on the side opposite to the abducens paralysis is usually present, owing to the physiological and anatomical association of the nerves supplying the rectus externus of one side and the rectus internus of the other. This form of alternate hemiplegia is accompanied by some conjugate paralysis, but close examination will show that paralysis of the abducens of one side predominates as a symptom over the loss of the power of conjugate movement.

FIG. 397.



Complete paralysis of the right third nerve; partial paralysis of left internal rectus (left divergent squint and limitation of movement inward); pupils unequal, the right nonresponsive to light, the left reacting to light and shade.

Hemiplegia with Irregular Varieties of Oculomotor Paralysis.—Instead of alternate hemiplegia of the ocular type, now and then hemiplegias with irregular varieties of oculomotor paralysis are

observed, as in the case shown in the illustration Fig. 397, which is an enlarged view of the face of the patient whose full length photograph is given in Fig. 308, page 574. This case was one of right hemiplegia with strong contractures involving the affected limbs, with marked atrophy in the upper extremity, with less complete paralysis of the right third nerve, and with limitation of the movements of the left eye. Dr. Charles A. Oliver has furnished me with the following ophthalmic examination of this case: "Vision with the right eye is lowered to one sixth of normal, that with the left eye being reduced to one tenth of normal. Accommodative power is very much lessened, especially in the left eye. The eye grounds are apparently free from any coarse changes. The pupil of the right eye is one third larger than that of the left eye, which is two millimetres in size. There is want of response of the right iris to light stimulus, with doubtful reaction of the left. The right superior and right inferior straight muscles are paralyzed. A slight action of the right external rectus muscle is obtainable. The right internal straight muscle almost entirely fails to act. The levator of the right lid is palsied, and the palpebral fissure is narrower than that of the fellow eye. The upper fibres of the right orbicularis muscle are less paretic than those of the corresponding lower portion. The lateral and vertical movements of the left eye are somewhat impeded, the excursions of the globe being accomplished by a series of coarse oscillatory motions. The left orbicularis muscle has good action." In this case the lesion was probably syphilitic and irregularly diffused or multiple,—a gummatous meningitis with some infiltration of the substance of the brain, one lesion being at about the superficial origin of the right third nerve, the other in the left half of the pons, so situated as to affect the pyramidal tracts and also some of the root fibres and association bundles of the ocular nerves.

Pareses or Paralyses of Ocular Muscles associated with Forms of Hemianesthesia.—Paresis or paralysis of one or more of the ocular muscles is sometimes found in association with hemianesthesia or even with partial bilateral anesthesia of the trunk and limbs. The anesthesia is usually of the dissociated type,—always in my experience partial or complete loss of the senses of pain and temperature. The most common combination probably is that of abducens paresis with loss or partial loss of pain and temperature sense on the same side. I have seen a number of such cases, with a clear syphilitic history, in which the patients had sudden attacks of vertigo, without unconsciousness, immediately after which they suffered from lateral diplopia with pathic and thermic hemianesthesia. These cases, under time and antisyphilitic treatment, usually greatly improve, but as a rule do not entirely recover. The diplopia sometimes disappears, the impaired sensation remaining, but in lessened degree. The syndrome would seem to be due to some form of focal lesion

affecting the substance of the pons, so as to invade the lemniscus and the abducens or third or fourth nerve root fibres, or the fibres which associate the ocular nuclei may be affected instead of the true root fibres of the nerves themselves.

Ocular Paralyses caused by Gross Lesions at the Base of the Brain.—A small gross lesion implicating any or all of the cranial nerves to the ocular muscles can be located with considerable accuracy in any one of several positions at the base of the brain. In doing this one of the chief dependences will be on the associated symptoms. Sometimes cranial nerves and encephalic tracts on both sides of the mesal line are evidently involved in the disease. Besides the nerves to the ocular muscles, the nerves most likely to be included are the ophthalmic division of the fifth, the optic, the olfactory, and the branches of the sympathetic which run in and near the cavernous sinus. The symptoms associated with a third, sixth, or fourth nerve palsy in the case of a gross lesion at the base of the brain will, therefore, usually be such as anesthesia in some of the subdivisions of the fifth, and especially in the distribution of its first branch ; vasomotor and trophic phenomena referable to implication of the fibres of the gangliated system ; partial or complete blindness from involvement of the optic nerve, and anosmia, parosmia, or some affection of smell from lesion of the olfactory nerve. In addition to these cranial nerve symptoms a hemiplegia from pressure upon the descending pyramidal tracts may be present. Both the internal and the external ocular muscles are, as a rule, conjointly attacked in such basilar lesions. Complete unilateral ophthalmoplegia is usually due to a gross lesion at the base. A case of this kind was studied by me in the Philadelphia Hospital. When first examined, the left eyeball was completely immobile and protruding, ptosis being almost complete. The pupil of the same eye was dilated and nonresponsive to light and to accommodation. Among the diagnoses suggested by different members of the staff were neuritis, gross lesion of the orbit, cavernous thrombosis, basic meningitis, aneurism or tumor at the base, and nuclear ophthalmoplegia. My own final diagnosis was thrombosis of the cavernous sinus with associated basic meningitis. Hearing and smell were impaired on the left side, and during the progress of the case the patient became totally blind in the left eye. Sensation to touch, pain, and temperature was at first abolished or greatly impaired in both the first and the second division of the fifth nerve. Under treatment with sodium iodide and mercurial inunction the pain and other active symptoms diminished, the area of anesthesia was reduced so as to be confined strictly to the distribution of the first branch of the trigeminus, and her mental condition improved. Coupland has recorded a similar but bilateral case with autopsy which showed a considerable basic meningitis, the third nerves being embedded in the exudation. The

cavernous sinus on each side was completely occluded by a thrombus of old date.

Ocular Paralyses due to Orbital Lesions.—Any or all of the muscles of the eye may be involved in an orbital neoplasm, abscess, or inflammatory disease of the orbit. The loss of movement may be dependent either on pressure or on the pain which results from such movement, or in part on both. A paralysis due to lesion of the muscle substance is rare, but edema at the insertion of the muscle or of several muscles may be present and will be accompanied by pain which may partially or completely inhibit ocular movements. Some poisonous drugs may paralyze ocular as they may other muscles. When the inferior oblique and internal ocular muscles of one side alone are affected, the case is not improbably due to an orbital lesion, for, as has been shown, an offshoot of that portion of the third nerve which goes to the inferior oblique passes to the ciliary ganglion, and, as the ciliary muscle and the iris are supplied from this ganglion, if the case is one in which the syndrome is paralysis of the inferior oblique with internal ophthalmoplegia and anesthesia, the lesion is probably orbital,—one involving the ciliary ganglion itself, and the nerve branches between it and the inferior oblique and internal ocular muscles. When an ocular paralysis is due to a tumor or an inflammation in the orbital cavity, exophthalmus will usually be present, and the optic nerve, as well as the nerves to the ocular muscles, will generally be involved. In time atrophy of the nerve, with partial or complete blindness, may result. In congenital oculomotor palsies the muscles within the orbit may be atrophied or absent, although in these cases the lesion may not be in a strict sense one of the orbit, but rather one of arrested development, the levator palpebræ and the superior rectus being the muscles the development of which is most frequently arrested. The orbital foramens offer opportunity for trouble by the growth of exostoses. Fracture is another cause of orbital paralyses.

Etiology.—*The Association of Disturbances of Ocular Movements with Degenerative Constitutional and Infectious Diseases.* In the consideration of the etiology and pathogenesis of the affections of ocular movements treated in the present chapter it is particularly necessary to keep in mind the frequent association of these disorders with various degenerative, constitutional, and infectious diseases. In a broad sense a discussion of the relations between these diseases and the disturbances of ocular movements constitutes a most important portion of the etiology of oculomuscular disease. Reference has already been made incidentally to some of these associations. Among these degenerative diseases are paralytic dementia, tabes, multiple sclerosis, Friedreich's ataxia, progressive muscular atrophy, and syringomyelia; and among important constitutional and diathetic affections are syphilis, tuberculosis, rheumatism, gout, lithemia, dia-

betes, and exophthalmic goitre. Diphtheria, scarlet fever, measles, influenza, and cerebrospinal fever are in this respect the most important infectious diseases.

Degenerative Diseases with Disorders of Ocular Movements. It will be remembered that Redlich investigated the occurrence of special disturbances of the direct and consensual iritic reflexes in paretic dementia (see page 822). The subject has claimed the attention of many other alienists and ophthalmologists. According to Bevan Lewis, the eye symptoms in general paralysis form a highly characteristic and significant group. Both the extrinsic and the intrinsic muscles suffer. The extraocular muscles present derangements in exceptional cases only, while the intraocular muscles are affected in some way in all cases at some stage of the affection. He summarizes the most frequent motor derangements as follows: (1) spastic myosis; (2) paralytic mydriasis; (3) all degrees of irregularity of pupil; (4) irregular contour from partial spasm or paralysis; (5) loss of sympathetic reflex; (6) loss of consensual movements; (7) reflex iridoplegia (Argyll-Robertson pupil); (8) associative iridoplegia; (9) cycloplegia. According to Oliver, the oculomotor symptoms of the third stage of general paralysis of the insane show paretic and paralytic disturbances connected with the oculomotor apparatus itself, all of greater amount and of more serious consequence than those seen in the same apparatus during the second stage of the disease. In a large percentage of cases of tabes some disorder of the ocular muscles is sooner or later present. The enumeration of these motor derangements occurring in paretic dementia would apply almost absolutely to tabes; probably cycloplegia is of less frequent occurrence in the latter. It is well known that a more or less transient diplopia is one of the frequent symptoms in the early stages of this disease. Examination may show no definite paralysis of any ocular muscle, while in other cases, even in the early stages, such paralysis, or an insufficiency which borders on a true paresis, may be readily determined. Instead of loss of power a true ataxia of the ocular muscles is occasionally observed. In one case recorded by Althaus the patient had the greatest difficulty in combining the ocular muscles to synergetic action. He easily saw double, and any sudden purposive movement of the eyes distressed him. It was exceedingly difficult for him to look upward, although the muscles concerned in this movement showed no trace of paralysis. Extensive investigations have been made upon the occurrence of affections of both the optic nerves and the ocular muscles in disseminated sclerosis. Uhthoff found from a study of one hundred cases of multiple sclerosis that paralysis of the ocular muscles, nystagmus, and nystagmic tremor may exist in this affection either separately or combined. Paresis of the ocular muscles was found in seventeen cases, and pronounced ophthalmoplegia externa in two instances, but in all the pupils reacted normally to light and

accommodation. In two cases upon convergence slight contraction was barely perceptible, and in a third case it was absent in one eye. Nystagmus and nystagmic tremor are of somewhat frequent occurrence both in tabes and in disseminated sclerosis. In Friedreich's ataxia a peculiar form of nystagmus is often one of the late symptoms. Friedreich believed this nystagmus to be due to disease of the ocular nuclei. It would seem more probable that it is dependent not only upon affection of the nuclei but on disease affecting the tract associating these nuclei together or uniting them with other parts of the brain. In progressive muscular atrophy both dilatation and contraction of the pupils have been observed. Commonly the contraction is unilateral, and it is probably dependent upon the degeneration attacking the ciliospinal centre. Ptosis, single or double, and other forms of extraocular paralysis have been recorded, but some of these cases may have been part of the syndrome of syringomyelia, which was formerly not clearly distinguished from amyotrophic lateral sclerosis and chronic muscular atrophy. A few cases of partial external ophthalmoplegia have been recorded as occurring in syringomyelia. The pupils are occasionally unequal. Iridoplegia and cycloplegia are not present. I have seen several interesting cases of syringomyelia with ocular and other bulbar symptoms. A man had had two apoplectic attacks, without loss of consciousness, six or seven years before. Lateral rotation of the eyes to the right and left was greatly restricted, and he had partial double ptosis. The arm and leg of the left side were paretic, as was also the right side of the face, particularly in its lower part. Irregularly distributed anesthesia to pain and temperature was present in both lower extremities. In this case a gliomatous process had probably attacked the lower portion of the spinal cord and the upper central region of the bulb. Nystagmus is a symptom of rare occurrence.

Constitutional and Diathetic Diseases with Disorders of Ocular Movements. Fully fifty per cent. of all cases of paralysis and spasm of the ocular neuromuscular apparatus can be attributed to syphilis, and among the forms of syphilitic disease which cause these disorders are gummatous meningitis, encephalitis, neuritis, and disease of the bloodvessels, such as nodose periarteritis. Syphilis exerts its influence upon the muscular apparatus both directly and indirectly, the symptoms sometimes being due to pressure, at other times to destruction or irritation or to both. The degenerative diseases which attack the oculomotor nuclei and tracts are in some instances parasyphilitic; in other words, they are the remote effects of syphilis which has set afoot processes which have determined the occurrence of degeneration. Tuberculous processes, and especially the basal form of tubercular meningitis, are often the direct causes of paralytic or spasmodic affections of the ocular muscles. Rheumatism acts chiefly as a peripheral cause, and sometimes attacks special branches

of single nerves, or even the muscles themselves, or the nerve endings in the muscles. When the external rectus or any other of the extraocular muscles is paralyzed apparently as the result of exposure, rheumatic neuritis or myositis may often be presumed. Rheumatic affections of the ocular muscles do not, however, usually present themselves during the acute rheumatic attacks of the articular variety. True gout sometimes originates ocular disorder, and meningitis or neuritis may involve the ocular as well as other nerves in its progress. Risley has called attention to the fact that lithemia is both a primary and a modifying factor in many ocular discomforts and disturbances. Paralyses of the extraocular muscles, and especially of the rectus externus, have been noted in a number of cases of diabetes. Oculomotor paralysis is occasionally seen in association with exophthalmic goitre. Usually both the internal and external recti are affected, or complete ophthalmoplegia may be present. Schlesinger has reported a case with exophthalmus, tachycardia, and enlarged thyreoid,—a typical case of Basedow's disease. When the patient extended her fingers they presented a fine tremor. When the eyes followed an object in an ascending plane the lids remained stationary, but not so in a descending plane. The right superior rectus muscle was paralyzed, an observation never before made. Recklinghausen has shown that in exophthalmic goitre a fatty degeneration of the muscles of the eye may occur. Other observers hold that the paralysis may be due to disease of the oblongata or of the peripheral nerves.

Disorders of Ocular Movements accompanying Infectious Diseases. Almost any form of infectious fever may be accompanied or followed by some type of ophthalmoplegia. The ocular muscles are particularly likely to suffer after diphtheria, and cycloplegia is the most frequent of the ocular disorders. The disorder of accommodation may be overlooked, especially in myopic patients, but it is of so marked a character in others, if they are old enough to use their eyes for reading or other forms of near vision, that it is readily recognized. Iridoplegia may be present, but is of much less frequent occurrence, and forms of external ophthalmoplegia are comparatively common. Of these the paralysis of the external recti is most frequent, and, as would be expected, this is often associated with paralysis of the ciliary muscle. Ptosis, total paralysis of all the muscles supplied by one third nerve, and almost complete paralysis of almost all the muscles of one side have been observed. (Gowers.) It is probable that not a few of the cases of diphtheritic paralysis of the muscles and ocular paralysis associated with other forms of infectious disease are due to forms of acute nuclear ophthalmoplegia, but if this is the case the inflammation is in most cases benign, as most of them recover. Diplegia and oculomotor paralysis have been observed after measles, cerebrospinal fever, influenza, and malaria.

Disorders of Ocular Movements of Toxic Origin. In the production of disturbances of ocular movements, poisonous substances, whether introduced from without or formed within the body, are related in their mode of action to constitutional and diathetic diseases, and especially to the infectious fevers. Their action, like that of these fevers, may be purely toxemic, they not having given rise to inflammation of either the nerves or the brain substance. Changes in structure and the symptoms which result may, in other words, be dependent, in many instances at least, upon the direct influence of the poisonous agent, or on a ptomaine or ptomaines generated in the blood. Alcohol, nicotine, lead, arsenic, carbolic acid, chloral, gelsemium,—in fact, almost any of the poisons which have been already discussed under the toxic amblyopias,—may be more or less influential in the production of the disorders of ocular movements. Gutmann reports a case of bilateral acute ophthalmoplegia following the use of spoiled meat, in which the paralysis affected all the external and internal muscles of the eye, and disappeared after two weeks. Other similar cases have been recorded. In the affection known as asthenic bulbar paralysis the ocular muscles are sometimes affected, ptosis with some involvement of the third or the sixth nerve being among the recorded symptoms. Although these cases are sometimes rapidly fatal, in some instances they are curable. The most reasonable explanation of the origin of this affection is the production in the system of poisons of exogenous or endogenous origin, these toxic substances exerting their most baneful influence on the ganglionic cells of the motor system, and especially on those of the bulbar region of the brain.

Congenital Ocular Paralyses. When speaking of bulbar paralysis of the sixth nerve, reference was made to the congenital origin of some of the forms of paralysis of the nerves supplying the ocular muscles. The exact pathological nature of these paralyses is not settled, or at least the lesions are probably different in different cases. It may be said in general terms that most of them are due to special forms of arrest of encephalic development: in most cases probably the nuclei are involved. In rare instances some of the ocular muscles have been entirely absent; in other instances they are inserted abnormally.

Affections of the Third Nerve in Migraine. It is well known that visual symptoms, and especially forms of spectra, are of frequent occurrence in attacks of migraine. Transient oculomotor paralyses also sometimes constitute a part of the symptomatology of this disorder. The paralyses are temporary, but often recur in the same patient at irregular intervals. Suckling, for example, reports a case of migraine followed by temporary paralysis of the third nerve, in a young man eighteen years old who had suffered since infancy with severe headaches, which gradually increased in frequency. The

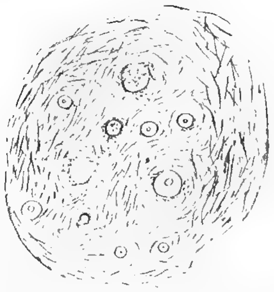
patient during his attacks had to remain in bed two days without sleep or food. The eye affection was first noticed in infancy, and had frequently recurred. The left eyelid began to droop when the pain was wearing off, and in twenty-four hours had completely dropped. On two occasions the eye had turned outward. This case is a type of a number of others that have been reported, although the ocular paralytic phenomena assumed somewhat different appearances.

Recurrent and Relapsing Oculomotor Paralysis. Interesting forms of transient oculomotor paralysis have been observed. One of these, of organic origin, can usually be traced to syphilis. Some or all branches of the third nerve may be involved, and in rare cases the fourth and sixth nerves may be implicated. The affection frequently attacks children; and in one of the cases reported local meningitis was discovered after death. Headache, nausea, and general malaise accompany the disorder. The cases are sometimes of rapid and at other times of slow development. Sometimes anesthesia and paresthesia of the face, trunk, or limbs are present. The cases of approximate recovery between the attacks are much more frequent than those in which the recovery is complete. Instead of complete recovery with recurrence it is perhaps better to speak of what usually happens as periodical exacerbations. Some of the organic cases are gliomatous rather than syphilitic; but, whatever their pathological origin, these cases must be carefully separated from those forms of transient oculomotor paralysis just considered which follow attacks of migraine. A few cases of recurrent paralysis of the third nerve with autopsy have been reported. It is of great interest to note that in all of these, in spite of the various theories advanced, some form of local disease of the nerve, as tubercle, fibroma, or meningitis, was found; but, as Jeffries remarks, in citing these cases they could not be called instances of true recurrent paralysis, since a certain amount of paresis had remained between the spells, but they were rather paresis of the third nerve with recurrent exacerbations. Gowers gives some interesting details as to the time of onset and methods of recurrence of some of these forms of ocular palsy. The attacks may begin in early childhood, and even in infancy, and may continue to middle life or even later. They may occur at about the same time each year, or at intervals of six months. No fixed limit, however, can be placed to the time of absence or recurrence, as in some instances they are exceedingly irregular. The attacks incline to be long when the intervals between them are long, and the reverse. The same authority describes a special form of *relapsing ocular palsy* which occurs usually in syphilitic subjects and has a particular tendency to relapse and persist. "One third nerve becomes paralyzed, improves under treatment, and then, perhaps while the treatment is being continued, the other third nerve suffers, and afterwards the affection of the first returns."

Ocular Paralysis of Hysterical Origin. Forms of ocular paralysis and spasm are sometimes observed in hysteria. Weir Mitchell has described a form of ptosis usually met with in women. He believes it to be of hysterical origin. In a large experience he had never encountered a case among men, and this accords with my own experience. Mitchell describes it as a "simple, quiet closing of the lids, and a resistance on attempting to lift them with the fingers, and also an absolute incapacity for a time to raise them by the will." This affection usually comes on in women who are neurasthenic or hysterical, and may have an emotional origin.

Miscellaneous Causes. Among causes not enumerated in the above paragraphs are injuries either directly to the nerves and muscles or involving the nuclei or trunks of these nerves at the base of the brain. In some instances fracture of the orbital walls has resulted in special forms of ocular paralysis. That some of the affections of ocular movement are due to disorder of refraction is now universally recognized, although perhaps too much stress is laid by some enthusiasts upon this etiology. Some cases of ocular paralysis not otherwise explicable are vaguely attributed to reflex disturbances, and it is possible that a focus of extreme irritation like a diseased and painful tooth may cause ocular paralysis by reflex inhibition, but such cases are, in my opinion, rare. A purely functional ophthalmoplegia is also very rare, and yet cases have been recorded which would seem to be capable of no other explanation.

Fig. 398.



Microscopical section of right oculomotor nerve, showing complete degeneration and almost total disappearance of the nerve fibres. High magnification. Specimen from case shown in Figs. 385 and 386.

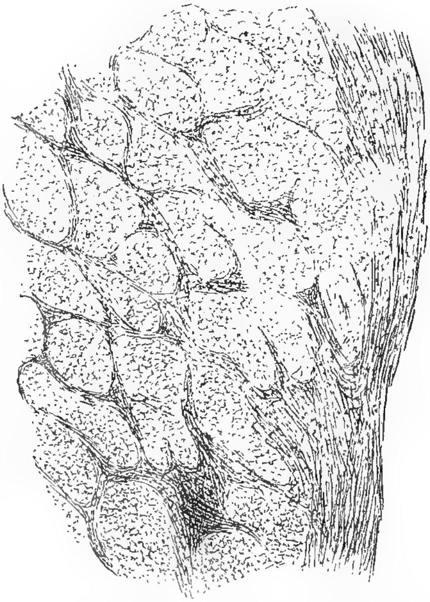
Fig. 399.

Microscopical section of right oculomotor nerve, showing complete degeneration and almost total disappearance of the nerve fibres. Low magnification. Specimen from case shown in Figs. 385 and 386.

Pathological Anatomy.—The pathogenesis and pathological anatomy of the disorders of ocular movements have been more or less fully considered in the discussion of special forms of these disorders in the previous pages. In syphilitic cases the lesions are

those which have been so often described as occurring in the course of encephalic syphilitic disease, no matter where it may be situated,—gummatous meningitis, focal or diffuse encephalitis, isolated or multiple neuritis, inflammatory disease of the large or small bloodvessels, and degenerative processes set up by the remote effects of the syphilitic virus. In one of my cases of third nerve paralysis almost the entire series of syphilitic findings were present. (Fig. 387, page 817.) Illustrations, drawn by Dr. Mary Alice Schively, are shown of the microscopical sections of the oculomotor and optic nerves, and of the gummatous formation with diseased vessels which was present in the Sylvian fossa. (Figs. 398, 399, 400, 401.) Some of the many changes which have been found in nuclear and peripheral cases may be summarized. Miliary hemorrhages have been present throughout the central gray matter of the third ventricle, the aqueduct, and the preoblongata. In some cases more or less extensive degeneration of the nuclei has been present, and this degeneration may be altogether similar to that which was found in chronic muscular atrophy. Gross lesions of all sorts have been discovered, as myelitis, tumor, and endarteritis. The well-known pathological conditions present in tabes, multiple sclerosis, and general paralysis have of course been noted. (Jeffries.) Of syphilitic disease of the membranes as a cause of eye paralysis Jeffries says that it is usually in reality a more or less diffuse gummatous or round cell growth springing from the dura and skull; and these growths, though small in size, can produce great mischief by creeping along the base of the skull and crowding the foramina. In the acute form of poliomyelitis superior hemorrhagica autopsies have shown in several cases numerous punctiform hemorrhages in the central cinerea. In one case, which lasted twenty days, commencing degeneration of the nuclei was found, being most advanced in the abducens and hypoglossus, less in the motor oculi, and least in the trochlear nerve. In the chronic progressive form of nuclear paralysis the changes usually consist in

FIG. 400.



Microscopical section of right optic nerve, from the case of third nerve paralysis shown in Figs. 385 and 386, showing new fibrous formation, round cell infiltration, and some degeneration of the nerve fibres.

atrophy and degeneration of the nuclei and roots, although some intact ganglionic cells are found in almost all cases. Ependymitis with secondary degeneration of the cinerea is another cause.

Fig. 401.

Microscopical section showing gummatous formation and caseation, endarteritis, and periarteritis, and involvement of the meninges and brain substance, from the case shown in Figs. 385 and 386.

Diagnosis.—While the subject of diagnosis is of great importance in connection with disorders of ocular movements, as elsewhere, little needs to be added to what has already been given under various heads. The methods of focal diagnosis have been detailed when discussing the symptomatology of lesions isolated to the different nerves supplying the ocular muscles, and of lesions of various points in the oculomotor pathway from the cortex to the orbit. As it is practically impossible to examine the ocular muscles electrically, we are shut out from this method of differentiating peripheral from central ocular paralysis, and must rely rather on the differences in symptomatology. The diagnosis of the nature of the lesion causing the ocular affection may be materially assisted by a study of the associated diseases and symptoms. When, for instance, oculomotor and oculopupillary phenomena are present in cases of tabes, multiple sclerosis, or paralytic dementia, the disease causing them is almost always of a degenerative type; and when they are due to lesions limited to peripheral ocular nerves or their nuclei, syphilitic lesions are their most frequent cause. The diagnosis of organic from functional or hysterical cases is usually not difficult, although recurrent forms of transient oculomotor paralysis may be due to syphilis, neurasthenia, or hysteria. When dependent upon the first of these causes, although the paralysis is transient and recurs it rarely disappears entirely; some evidences of its presence can nearly always be discovered on careful examination. The ocular type of asthenic bulbar paralysis

can scarcely be diagnosticated with certainty at an early period. It will be remembered that it comes on usually after feelings of great fatigue and rapid exhaustion in all the parts which are eventually affected, and that no constitutional or other cause can be determined. But this subject will be more fully treated later. Ocular palsies and spasms of hysterical type are to be differentiated chiefly by a search for the stigmata of hysteria. It needs only to be remembered that hysteria and organic disease of the encephalon may be present in the same case. The absence of the pupil reflex is usually, and I believe correctly, regarded as significant of organic disease, although Karplus called attention to its absence in what he believed to be attacks of hysteria, and Feré seems to have noticed this before. Karplus's observations were made in the clinic of Prof. Krafft-Ebing, who confirmed the diagnosis and the fact of the absent reflex. The latter was also confirmed by the ophthalmologist Bernheimer. The observations were made by having the lids held apart, the eye being illuminated by a hand lamp and the cornea protected by a salt solution. Thus the eyes could be observed for several minutes. The pupils were wide open and motionless for twenty seconds. These observations, if confirmed, will make it necessary to use with reserve the absent pupil reflex as a negative pathognomonic sign in suspected hysteria. It is necessary to be on one's guard against accepting too readily the statement that the absence of the pupil reflex in this case was purely an hysterical phenomenon. A nuclear degeneration or a toxine acting upon the nucleus for the sphincter iridis might have been the cause of the phenomenon. "To make the diagnosis of paralyses of the ocular muscles, it is not sufficient to know the signs of the paralysis of each individual muscle and then see to which of them any case that may be before us fits. In this way, to be sure, we would quickly make the diagnosis in the typical and uncomplicated cases, but in the numerous cases of combined paralysis we would be helpless. A much more proper way of going to work is to determine exactly all the symptoms in any given case, and from them find out in what direction the motility of the eye is incomplete." (Fuchs.)

Prognosis.—The prognosis of disorders of ocular movements varies according to the nature and the site of the lesion. Cases occurring in the course of degenerative diseases are necessarily unfavorable in prognosis, as they are portions of a disease which tends to tissue death. Cases which are due to syphilis vary in prognosis according to the nature, extent, and destructive influences of the lesion. Gummatous meningitis gives a comparatively favorable prognosis, although when the meningitis is associated with infiltration of the brain substance recoveries are usually only partial. When the lesion causing the ocular affection is an embolus or a thrombus the prognosis is unfavorable, at least so far as the persistent affection is dependent upon the destructiveness of the lesion;

but in some of these cases, as in other affections due to embolism and thrombosis, the early symptoms are more pronounced and extensive than those which persist, a fact which is of course explained by the acute conditions which are present at first and which disappear in whole or in part as the result of the establishment of collateral circulation. The prognosis in diseases like tubercular meningitis is simply that of the general affection. The discussion of recurrent and relapsing cases has sufficiently indicated their prognosis. In infectious and toxic cases and in those due to neurasthenia, or those which form a part of the syndrome of hysteria, the prognosis is good. Cases due to neuritis, whether syphilitic or rheumatic, afford a comparatively favorable prognosis, as do also those associated with affections like gout and lithemia.

Treatment.—Strychnine or nux vomica is the most valuable of stimulating drugs in the treatment of oculoparalytic affections. It should be given in full or augmenting doses, and may be used by the mouth or hypodermatically. In some cases the hypodermatic use of strychnine nitrate appears to be of great service, as it is in optic nerve atrophy. In rheumatic cases, in the acute stage the salicylates and such remedies as the potassium and sodium carbonates may prove of great service, and pilocarpine and other diaphoretic drugs may also be of value. Hysterical cases are to be treated by methods applicable to hysteria of any form,—by tonics and nutrients, by static electricity, and by various forms of suggestion. Even hypnotic suggestion may here be useful. Syphilis attacks the neuromuscular apparatus in a variety of ways,—through disease of the blood-vessels which supply its centres and peripheral portions, through tumors, through gummatous meningitis, and by arousing into activity processes which eventually lead to degeneration. A consideration of these different methods in which syphilis acts on the nervous system will be helpful in the details of treatment. If the patient is believed to be suffering from a recent and more or less acute lesion, such as a neuritis of the third, sixth, or fourth nerve, or an active gummatous meningitis, specific treatment should be pushed heroically. The patient should at once be put upon the hypodermatic use of mercury, or on inunctions, after the method already described (see page 225). As soon as constitutional effects are produced, this treatment should be emphasized by the use of the iodides and hydriodic acid. If the lesion is of older date and more organized,—a gumma or a fibroid meningitis, for instance,—then both mercurial inunction and the iodides should be actively pushed. If the lesion is one of a destructive character, the indirect result of disease of the vessels, due, for instance, to thrombosis or endarteritis of terminal vessels, some good may be accomplished by the persistent but moderate use of antisyphilitic treatment, but the destructive portion of the lesion will remain in spite of any treatment, which will only retard future

and more rapid inroads on the vessels. If the disease, although due to syphilis, is of degenerative type, specific treatment will be of no avail. Such cases are helped most by tonic and nutrient treatment. When it is impossible to remove diplopia, and when the double images cannot be discarded by the patient, various means may render this condition more bearable. In rare cases prisms can be used to fuse the images, but in true ocular paralysis this will usually not succeed. The affected eye may be covered with a patch or a piece of ground glass, or an opaque disk of any kind may be put in the spectacle frame over the affected eye. The question of operative procedure in cases of ocular paralysis, insufficiency, and spasm is one that belongs almost exclusively to the ophthalmologist, but in neurological practice it is sometimes necessary to give opinions as to the propriety of such procedures and to differentiate the cases in which they should or should not be resorted to. Some of the questions presented for decision are such as the following. It is first necessary to decide whether the insufficiency, paralysis, or spasm is due to intracranial lesion or to errors of refraction. If due to the latter, in some cases partial operations or progressive tenotomies are desirable, but these cases are not so numerous as their enthusiastic advocates would have us believe. An experience extending over many years has led me to this conclusion, although I am convinced that in a comparatively limited number of cases tenotomies and partial tenotomies are useful in relieving the nervous and other symptoms dependent upon eyestrain. If the affection of the ocular muscles is dependent on an intracranial lesion, the question arises whether an operation can do any good. In rare cases tenotomy may be of transient service, but as a rule operation should be avoided, as it should be likewise in cases of degenerative disease with associated ocular disturbances. The suspension treatment used in tabes and disseminated sclerosis is said to have resulted in relief of the ocular symptoms. Michel has suggested a mechanical treatment to stimulate the weakened muscles, in practising which the eyeball is drawn backward and forward with force in the direction of the ordinary action of the affected muscle. Simple massage of the affected muscle has also been recommended. A galvanic current strong enough to reach the ocular muscles by direct application will most likely prove too irritating to either the external structures of the eye or the retina or to both. Possibly some good may be done in cases of partial paralyses of the peripheral type by carefully using very weak galvanic currents or faradic currents. A very small electrode covered with clean absorbent cotton which has been dipped in pure sterilized warm water should be applied over the muscles affected, the other large electrode being placed almost anywhere, as at the back of the neck or at the side of the face. Another method is to apply one electrode over the closed lid.

CHAPTER IX.

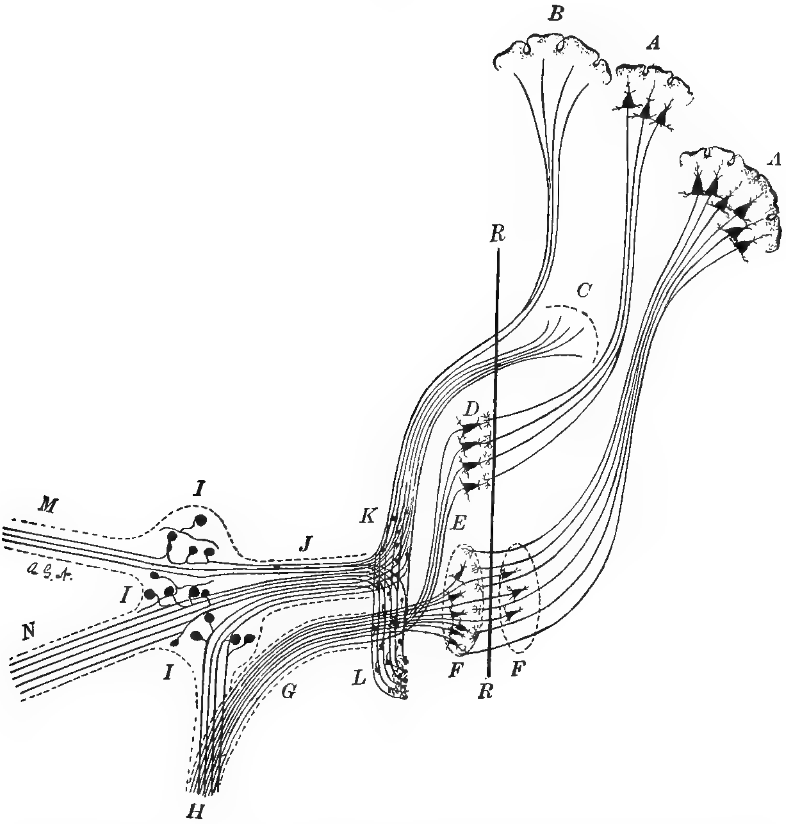
DISEASES OF THE TRIGEMINAL AND FACIAL NERVES, AND SMALL GROSS LESIONS OF THE PONS AND THE PREOBLONGATA.

ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS RELATING TO THE TRIGEMINUS.

General Description of the Trigeminus.—The largest of the cranial nerves is the trigeminus (*trigeminal nerve, trifacial nerve, fifth nerve*). It is a mixed nerve, having a motor portion (*portio minor*), and a much larger sensory portion (*portio major*). It is most in accord with recent investigations and the newer anatomical and physiological methods to consider separately the diseases of the sensory and the motor portion of the trigeminus, and they will in the main be discussed apart in different sections of the present chapter; but it must not be overlooked that at and near their origins gross lesions may affect both, although this rarely occurs. A lesion, for instance, at the so-called "superficial origin" of the nerve,—which is in reality only the superficial origin for the motor portion, it being the point of entrance to the brain stem of the sensory portion,—if of sufficient size, will cause symptoms both motor and sensory; and similarly a lesion might be so situated as to affect both the motor and the sensory nuclei or their intraencephalic or extraencephalic roots, including in the latter the Gasserian ganglion. Outside of the skull the motor root joins the inferior maxillary division of the sensory portion of the nerve, and again at this point a lesion might give symptoms of both a sensory and a motor character in the distribution of this division of the nerve. The anatomy of the trigeminus is of great complexity, both in its subdivisions and in its relations to other nerves and important structures. The distribution and relations of its radicles are, as will presently be seen, extensive and complex. The larger, sensory portion of the trigeminus is usually described as passing into the Gasserian ganglion, but it should be spoken of rather as springing from this ganglion, which is its true source, its nucleus of origin. The motor part arises in a chief motor nucleus situated in the metepicelian floor. It probably also receives most or all of the fibres which pass downward from the so-called *accessory* nucleus in the mesencephalon (in the aqueductal cinerea beneath the quadrigemina). The single axis cylinder processes of the neurons which originate in the Gasserian ganglion bifurcate, sending one set of their processes brainward to enter the lateral aspect of the pons, ramifying in the sensory nucleus of the pre-

oblongata, some of the fibres passing downward to the superior cervical cord, and others ascending. The other branches of the bifurcating Gasserian processes pass peripherally to form the different sensory divisions of the nerve,—the first, or ophthalmic, the second, or superior maxillary, and the third, or inferior maxillary division. The great cell nests and roots of the trigeminus are shown in Fig. 402. The central neuron of the motor trigeminus arises in

FIG. 402.



Scheme of trigeminal apparatus: *A*, *A*, cortical centres for trigeminal movements; *B*, cortical terminus of the trigeminal sensory tract; *C*, thalamus to which the central trigeminal sensory tract may be in large part distributed; *D*, accessory (motor) nucleus; *E*, descending (mesencephalic) root; *F*, chief motor nucleus; *G*, motor roots; *H*, inferior maxillary nerve; *I*, Gasserian ganglion; *J*, sensory roots between the Gasserian ganglion and the pons; *K*, ascending sensory root; *L*, descending (spinal) root; *M*, first or ophthalmic division of the trigeminus; *N*, second or superior maxillary division.

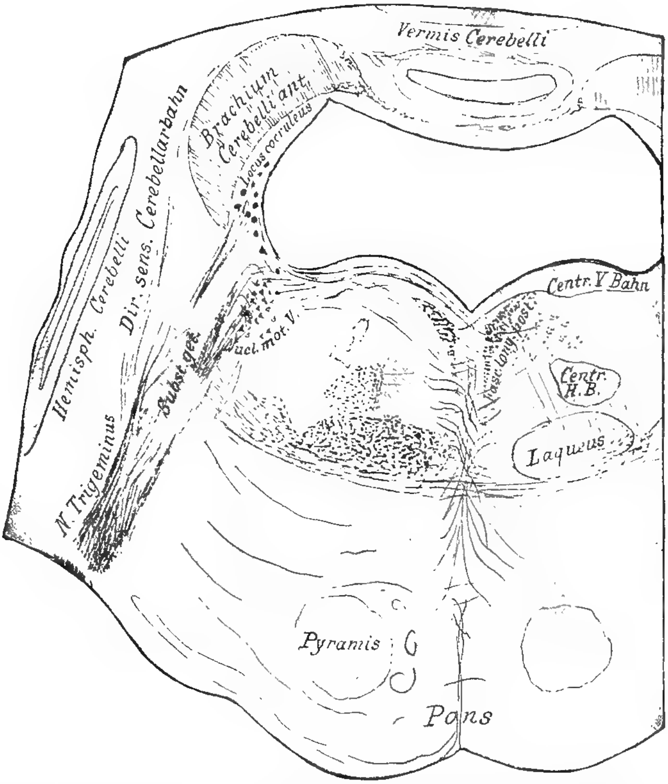
the cerebral cortex, probably in the lower extremities of the central convolutions, just behind the centres for laryngeal and pharyngeal representation, and near the centres for the lips and tongue (Fig. 228, page 333); its downward course is not certainly known, but it

probably passes about the knee of the capsule behind the fasciculus for eye movements (Fig. 236, page 360), separates in the crusta from the bundle of Spitzka, decussates in the tegmental raphe, and terminates in the cells of the chief motor nucleus in the preoblongata. The peripheral motor neuron passes from this nucleus by way of the motor root of exit, eventually to become a part of the inferior maxillary or mandibular nerve.

Sensory Portion of the Trigeminus.—*Sensory Roots.* The true nuclei of origin of the sensory portion of the trigeminus, as stated in the last paragraph and as shown in the diagram Fig. 402, are in the monopolar cells of the Gasserian ganglion. The large extraencephalic sensory root of the fifth nerve is constituted by the union of the delicate brainward passing branches of the processes of these cells. Each of the fibres of this sensory root bifurcates after entering the pons, one branch ascending and the other descending, as first described by Ramón y Cajal. Van Gehuchten believes that the ascending branches of the sensory root pass into the descending motor root of the fifth, and with the latter to the postgeminum. This Ramón y Cajal does not accept. The ascending branches of the sensory root, according to the latter, terminate in free ramifications in the substantia gelatinosa. Although these fibres are extremely fine, from two to four collaterals are given off at right angles from them, and these, ramifying between the cells of the substantia gelatinosa, form a thick plexus. The long and large spinal root of the trigeminus was formerly regarded as ascending from the spinal cord; but it is now known that it is composed of the very numerous delicate sensory fibres which descend after their entrance and bifurcation in the pons. Compared with the sensory fibres which take an ascending course, these descending branches are thick. They may be traced downward below the pyramidal decussation into the cord itself about as far as the second cervical segment, and it is possible that some fibres descend still farther. This is an important fact, and it is probably due to this that sensation of the face is sometimes affected in lesions of the upper cervical region, as in syringomyelia. About the level of the decussation they occupy the place which Lissauer's zone has lower down, being directly lateral and dorsal to the substantia gelatinosa. This descending fifth root has a semilunar form, and consists of two layers, a superficial one, formed of coarse fibres, and a deep layer, containing vertical fibres which are separated by nerve cells and collaterals. The fibres of both give origin to collaterals which ramify in the substantia gelatinosa. The portion of the substantia gelatinosa in connection with the spinal root is its end nucleus, and accompanies it throughout its course. The cell nest which has been called the sensory fifth nucleus is merely the better developed pontile portion of this end nucleus. Collaterals from this spinal root pass in animals to the cell nests of the motor cranial nerves. From the end nucleus of the

fifth arise fibres which mingle with the fillet fibres, cross the raphe, and ascend in the tegmentum to the brain. The axis cylinders of the

FIG. 403.



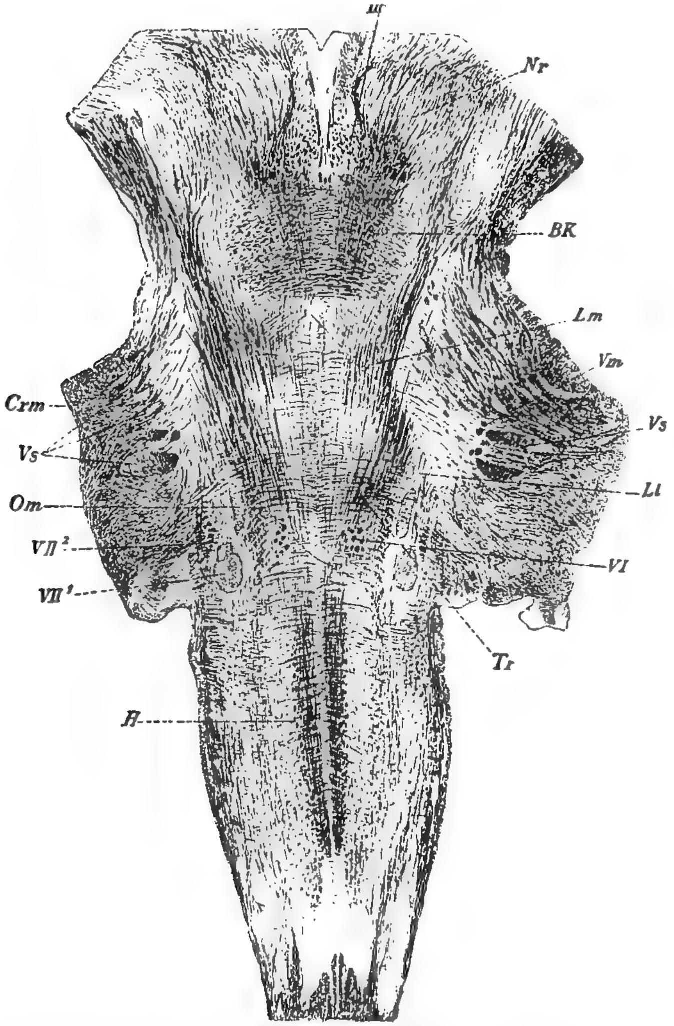
Section of the pons at the point where the descending (spinal) root of the trigeminus bends outward: *Centr. H. B.*, central tegmental tract; *Centr. V. Bahn*, central tract of the trigeminal nerve; *Dir. sens. Cerebellarbahn*, direct sensory cerebellar tract; *Laqueus*, lemniscus or fillet. The other structures represented in the illustration are indicated by their well known Latin names. (Edinger.)

cells within the substantia gelatinosa constitute the central sensory tract of the trigeminus. These cells are divided into large and small, and the nerve processes, which have here no collaterals, probably pass mesad to cross the raphe and then bend, taking a longitudinal direction to form this central sensory tract. (Koelliker and Ramón y Cajal.) The position, course, and relations of the sensory and motor roots of the fifth nerve are well shown in Fig. 404. Fibres pass from the sensory fifth root laterad of the prepeduncle of the cerebellum (Edinger's sensory cerebellar tract, Fig. 403).

Peripheral Course of the Sensory Portion of the Trigeminus. The first or ophthalmic division of the fifth nerve passes along the outer wall of the cavernous sinus, in close relationship with the third and sixth nerves and with the ophthalmic artery and vein, dividing into

three branches, the frontal, the lachrymal, and the nasal. The frontal is the largest of the three branches, and subdivides into the supra-orbital and the supratrochlear. The entire ophthalmic nerve passes

Fig. 404.

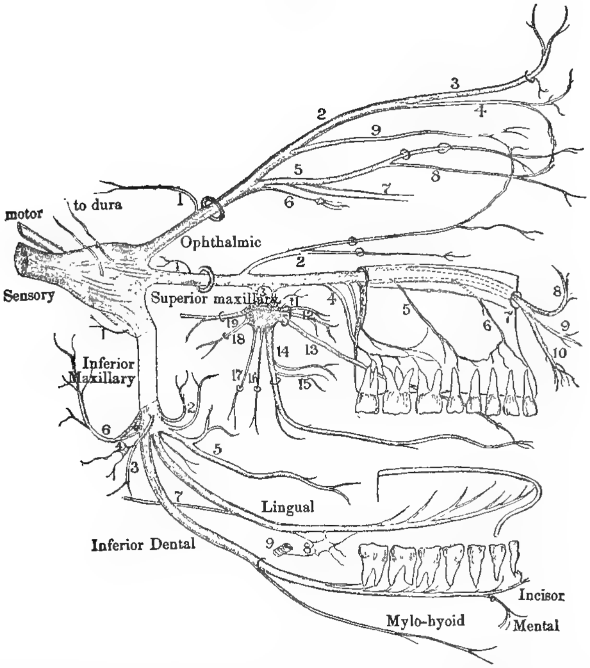


Frontal section of the preoblongata and pons of man: *H*, hypoglossal roots; *VI*, abducens; *VII*¹, facial nucleus; *VII*², facial root fibres; *Vs*, sensory roots of fifth nerve; *Vm*, motor roots of fifth nerve; *III*, oculomotor root fibres; *Tr*, trapezium; *Om*, superior olive; *Lm*, median lemniscus; *Ll*, lateral lemniscus; *Crm*, cerebellar peduncle; *BK*, decussation of the prepeduncles; *Nr*, red nucleus. (Koelliker.)

from the skull into the orbit through the sphenoidal fissure. The second or superior maxillary division leaves the Gasserian ganglion, proceeds through the foramen rotundum, and then takes a horizontal course forward through the sphenomaxillary fossa; next it passes

under the orbit and through the inferior orbital canal, emerging on the face at the infraorbital foramen. The third or inferior maxillary division, leaving the Gasserian ganglion, passes downward through the foramen ovale to emerge with the motor subdivision at the base of the skull. The posterior or great sensory subdivision of this nerve divides into three branches, the auriculotemporal, the lingual, and the inferior dental. The numerous branches of the three great divisions of the fifth nerve are sufficiently indicated in the diagram with its legend (Fig. 405).

FIG. 405.

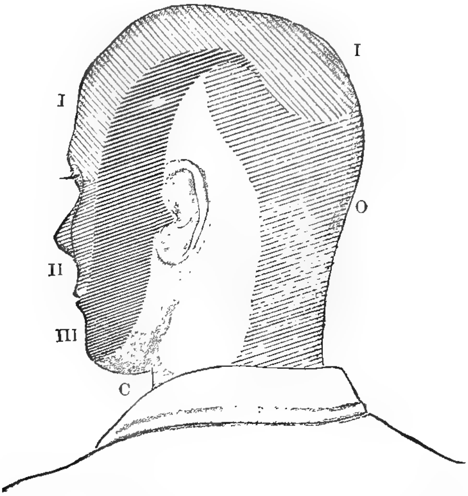


Branches of the fifth or trigeminal nerve. The main roots and subdivisions of the nerve are named on the illustration. Ophthalmic division : 1, recurrent branch to dura ; 2, frontal ; 3, supraorbital ; 4, supratrochlear ; 5, nasal ; 6, branch to ciliary ganglion ; 7, long ciliary branches ; 8, infratrochlear ; 9, lachrymal. Superior maxillary division : 1, recurrent branch to dura ; 2, orbital or temporomalar ; 3, Meckel's (sphenopalatine) ganglion ; 4, posterior dental ; 5, middle or external dental ; 6, anterior dental ; 7, infraorbital plexus ; 8, palpebral ; 9, nasal ; 10, labial ; 11, ascending branches of sphenopalatine ganglion ; 12, superior nasal ; 13, nasopalatine (nerve of Cotunnius) ; 14, anterior palatine ; 15, infranasal ; 16, middle or external palatine ; 17, posterior or small palatine ; 18, pharyngeal ; 19, Vidian. Inferior maxillary division : 1, recurrent branch to dura ; 2, muscular ; 3, pterygoidal ; 4, otic ganglion ; 5, long buccal ; 6, auriculotemporal ; 7, chorda tympani ; 8, submaxillary ganglion ; 9, facial artery. (Hughes.)

Trigeminal Cutaneous Areas. A knowledge of the cutaneous areas to which the trigeminal sensory nerves are distributed is of importance to the clinician. The general areas for the first, second, and third divisions and also for the anterior division of the second cervical and for the occipital nerves are shown in Fig. 406. The main

subdivisions of these great nerve branches to the face are shown in Fig. 407.

FIG. 406.



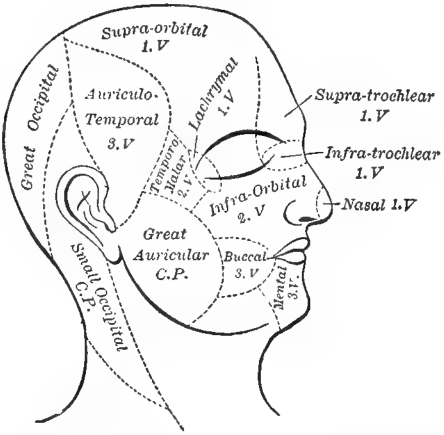
Cutaneous sensory areas of the trigeminal nerve: I, first or ophthalmic division; II, second or superior maxillary division; III, inferior maxillary division; C, anterior division of the second cervical nerve, which supplies chiefly the front and side of the neck. The small occipital nerve (*occipitalis minor, occipitalis parvus, occipitalis externus, occipitalis anterior*), which supplies the skin behind the ear and the area directed in a pointed shape towards the crown of the head, is not indicated by shading or lettering. O, great occipital nerve (*occipitalis major, occipitalis magnus, occipitalis maximus, occipitalis internus*), representing the posterior branch of the second cervical nerve, which supplies the skin corresponding with the upper part of the occipital bone and adjoining part of the scalp, as shown by the shading which extends towards the vertex. A branch of the posterior division of the third cervical nerve also ramifies in the skin above the occipital protuberance, and the pneumogastric has an auricular branch which appears cutaneously behind the ear.

Physiological Observations.—*Experiments on the Descending (Spinal) Root.* Turner, experimenting in connection with Ferrier, found that the results of a destructive lesion localized in the tubercle of Rolando on one side were unusually constant. Destruction of this tubercle caused loss of all forms of sensibility in the distribution of the trigeminus on the side of the lesion, this anesthesia being due, according to Turner, to the destruction of the great descending spinal root, where it forms the superficial layer of the Rolandic tubercle. As regards the body and limbs, the experiments seemed to cause loss of tactile sensation on the side of the lesion and of the pain sense on the opposite side. Other results of the experiments were, on the same side as the section, contraction of the pupil, temporary narrowing of the palpebral fissure and less prominence of the eyeball, and paresis of the arm and leg of the opposite side. A few experiments practically confirmatory of the results of Turner have been performed by others.

Trophic Functions of the Trigeminus. Somewhat numerous physiological experiments and a very few clinicopathological observations

indicate that in some way trophic phenomena are induced by lesion of the fifth nerve. Turner in a series of experiments performed by Ferrier and himself divided the trunk of the fifth nerve, its ophthalmic branch, and its intramedullary roots. In eighteen of the experiments anesthesia of the cornea was a prominent symptom, but only two of these showed symptoms of inflammation and destructive change, although in many of the cases a slight corneal opacity which showed no tendency to progress was produced. This, Turner believes, may have been due to nonapproximation of the lids and consequent drying of the corneal surfaces. Only one case in which the ophthalmic branch was divided showed distinctive corneal change, and in this case postmortem evidence of septic meningitis was found. Several of the experiments seemed to demonstrate that, whether the trunk of the nerve was divided behind the Gasserian ganglion or whether the section was of the ophthalmic branch alone, the processes of health, nutrition, and repair went on notwithstanding the anesthesia of the cornea. The general conclusions drawn were that there were no evidences of trophic influence exerted by the Gasserian ganglion upon the cornea, that the destructive changes in the cornea which occur from intracranial lesions are due to inflammatory irritation of the nerve, and that the so-called neuroparalytic phenomena associated with lesion of the trigeminal nerve are evidences of irritation of the nerve, and not of paralysis. The operations on the Gasserian ganglion have, on the whole, confirmed the view taken by Turner. According to Krause, for example, after such operations trophic diseases of the eye or of the skin do not, as a rule, follow, although in a few instances slight changes in the skin have been observed. The two well known cases of hemifacial atrophy reported by Homén and Mendel (see page 887) point to a trophic function for the fifth nerve. While the exact part played by the trigeminus in the control of trophic phenomena must be regarded as still unsettled, the clinician should remember that irritative lesions of various portions of the trigeminus may cause trophic symptoms.

FIG. 407.



Normal distribution of the trigeminal nerve to the face, showing the subareas of its three great sensory divisions. (Flower.)

Ophthalmic Herpes Zoster and Neuroparalytic Ophthalmia. Many

cases of ophthalmic herpes after trigeminal lesions have been recorded. Sometimes this disease is of so severe a character as to cause the destruction of the eye, vesication, pustulation, and ulceration of the cornea taking place. The patient suffers from darting pains about the orbit and in the eye. Perforation of the cornea, cyclitis, and shrinking of the eyeball have occurred. Anesthesia is present in the entire ophthalmic branch of the fifth nerve. Herpes zoster, wherever located, is usually due to disease of the ganglia in which the sensory nerves originate. In at least one case of intercostal herpes, to which reference is made by Lloyd from the personal observation of Dr. William G. Spiller, who saw the case in the service of Kovacs, in Vienna, disease was found of the dorsal spinal ganglion of the nerve along which the course of the herpes occurred. The case was one of Pott's disease in the cervicodorsal region, and at the autopsy miliary tubercles were found covering the ganglion. The ganglion above the one involved in the tuberculous process was intact, and no herpes was present in the tract of its nerve. In other cases of herpes dilatation of the vessels of the posterior roots at or near the points where they enter the cord has been noted. In neuroparalytic ophthalmia, another well-known trophic affection of the fifth nerve, inflammation, acute or chronic, attacks the eye and often results in its destruction. Vesication, ulceration, sloughing, and even perforation of the cornea may take place, the iris may become involved, and eventually the patient suffer from a panophthalmia. Anesthesia is commonly present in the conjunctiva, the cornea, and other parts supplied by the ophthalmic branch of the trigeminus. The case of combined facial and trigeminal paralysis whose photograph is shown in Fig. 420, page 914, suffered from a trophoinflammatory affection, and it became necessary to remove the affected eye, which was done by Dr. de Schweinitz. The lesion in this case probably involved the nuclei or root fibres of the trigeminal nerve. Kalt has reported that certain slight trigeminal alterations which produced hemifacial atrophy also caused central choroiditic lesions, and he believes that these results are due to an interference with the trophic functions of the trigeminus. He had also several times observed that central corneal opacities, interstitial and without erosions or cicatrices, came on slowly in persons who had suffered only from periorbital neuralgia. The centre of the cornea where the opacity is usually encountered is alone anesthetic, or at most the anesthesia extends in a minute circle of transparent tissue around it. A similar anesthesia is sometimes observed after operations for the correction of strabismus, and may involve a large part of the cornea without producing other effects than a slight temporary loss of epithelium.

The Motor Portion of the Trigeminal Apparatus.—*The Chief Motor Nucleus.* This nucleus, the *masticatory nucleus* of some authors,

is situated in the pons, mesad to the sensory nucleus of the trigeminus. The cells of the motor nucleus are stellate, and, unlike those of the accessory nucleus, have several processes. Their axis cylinders pass downward without collaterals, close to the sensory root, and take their exit from the pons near it. Ramón y Cajal, although he saw collaterals of the sensory root entering this nucleus, could find no connection of the pyramidal tract with it, and he has observed no decussation of the motor fibres. A large number of root fibres from the substantia ferruginea unite with the mesencephalic root of the fifth and pass to the motor root, although many deny the relation of this structure to the fifth nerve. Some authorities believe in a partial decussation of the motor roots, so that each root arises in both nuclei. Through the connection of the motor nucleus with the sensory root of the fifth the reflex movements of the muscles of mastication are possible. The motor nucleus is connected by collaterals of the dorsal longitudinal bundle with parts below it even as far caudad as the cord.

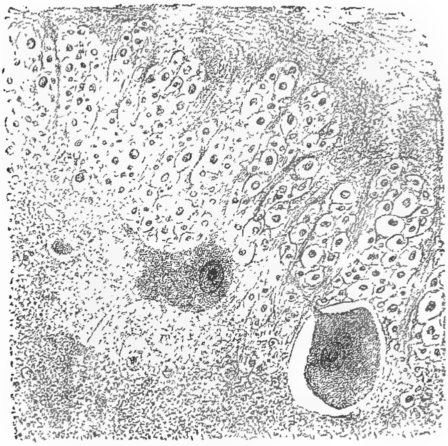
The Accessory Nucleus and Descending (Mesencephalic or Cerebral) Root. The vesicular cells which form this nucleus are spherical or pear-shaped, and provided with thick spines. Golgi, Lugaro, and Ramón y Cajal believe that they are unipolar, but according to Koelliker they are multipolar. In the adult animal these cells probably contain no processes. They form a column which begins at the quadrigeminum close to the prepeduncle and becomes thicker nearer the chief nucleus. Their axis cylinder processes give origin to numerous ramifying collaterals. They do not cross the raphe. This descending root of the fifth is also spoken of under various names, as the *small root*, the *cerebral* root, the *mesencephalic* root, the *superior* root, and the *anterior* root. As the long root is now also properly spoken of as "descending," it would perhaps be better to designate the great root as spinal, and the root of the accessory nucleus as mesencephalic.

Peripheral Course of the Motor Portion of the Trigeminus. Arising from the motor chief nucleus, the motor root passes forward with the sensory root through an oval opening in the dura on the superior border of the petrous bone above the internal auditory meatus. At the petrous apex it passes beneath the Gasserian ganglion, with which it has no connection. Outside of the cranium it joins one of the distal or peripheral bundles of the Gasserian ganglion, to form with it the inferior maxillary division of the fifth nerve. (Fig. 405.)

Muscles supplied by the Motor Division of the Trigeminus. First and chiefly the motor portion of the fifth nerve supplies the most important muscles of mastication, namely, the masseters, the temporals, and the external and internal pterygoids. The branch to the internal pterygoid muscle is connected at its origin with the otic ganglion, while that to the external pterygoid is most frequently de-

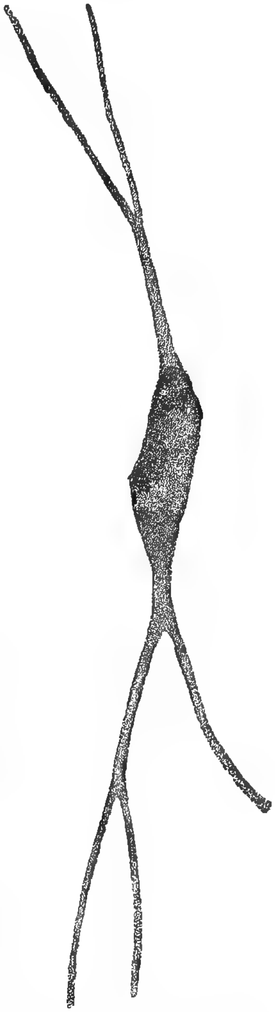
rived from the buccal nerve, although it may be given off separately from the interior trunk of the nerve (Gray). Besides the main mus-

Fig. 408.



Microscopical preparation from the descending (mesencephalic) root of the fifth nerve. In this preparation alongside the transverse sections of the root fibres two cells are represented, one with four processes, and a second one which has but one recognizable process. (Koelliker.)

Fig. 409.



Enlarged view of one bipolar cell of the descending (mesencephalic) branch of the fifth nerve. (Koelliker.)

cles of mastication, others which take some part in the processes of chewing and deglutition are supplied by the trigeminus. The mylohyoid muscle, for instance, is supplied by the mylohyoid branch of the inferior dental nerve, and the anterior belly of the digastric by the terminal mylohyoid filaments. The mylohyoid muscle assists in raising the tongue, the floor of the mouth, and the mylohyoid bone, as in mastication and the initial stage of deglutition. The anterior belly of the digastric muscle, acting from above, pulls the hyoid bone upward and forward, and acting in the opposite direction it assists in depressing the lower jaw. These muscles, acting in conjunction with others, are therefore important in the process of mastication. According to an old view, the nerve supply to the tensor palati is from the trigeminus, although it is usually assigned to the vagus. If derived from the former, the nerve branch passes in the vagus trunk, leaving it to go by way of the otic ganglion to the muscle. As the tensor palati acts to tighten the soft palate and open the Eustachian tube during deglutition, it is not

improbable that its nerve supply would be from the same source as that for some of the other muscles of mastication. The symptoms present in one of my own cases of combined paralysis and spasm of the trigeminus (Fig. 415) indicated the probable trigeminal origin of the accessory muscles of mastication just described. Finally, the tensor tympani muscle, which tightens the membrane of the tympanum, has its nerve supply from the otic ganglion, probably by a filament which is derived from the motor portion of the fifth nerve.

DISEASES OF THE SENSORY PORTION OF THE TRIGEMINUS.

Varieties of Trigeminal Sensory Disease.—Diseases of the sensory portion of the fifth nerve are, in the first place, divided into those in which sensibility is abnormally increased (*hyperesthesias* and *hyperalgesias*) and those in which sensibility is lowered or lost (*anesthesias*). Under the affections of hypersensibility are included (1) *symptomatic trigeminal neuralgias* (painful trigeminal affections due to irritative focal lesions, to neuritis or to special infections or toxins acting on the trigeminal apparatus); (2) the severe paroxysmal type of trigeminal neuralgia known as *prosopalgia* or *tic douloureux;* (3) *migraine* and *migrainoid neuralgias*, which are probably best treated of under trigeminal diseases, although much diversity of opinion exists as to their real nature. Certain forms of painful disease in the distribution of the trigeminus are classified under the head of trigeminal reflex disorders. These include varieties of *reflex headaches*, although some of these affections classified as reflex are more properly to be regarded as diseases due to direct irritation of the branches of the trigeminus. The most important disorders of lowered sensibility (*trigeminal anesthesia*) are (1) trigeminal anesthesia due to gross focal lesions; (2) trigeminal anesthesia due to degenerative disease attacking the nuclei and the root fibres of the nerve; and (3) trigeminal anesthesia due to chronic retrogressive processes like gliosis, which attack the trigeminal apparatus in the brain stem. *Hemifacial atrophy* and *hemifacial hypertrophy* are trophoneuroses, symptoms of sensory irritation not being prominent, although sometimes present. With our present knowledge, they are most conveniently treated of as affections of the sensory portion of the trigeminus, the weight of opinion, as will be seen later, being in favor of the view that the trophic phenomena of these diseases are due to neural or nuclear irritation. Instead of true hyperesthesias and anesthesias, forms of trigeminal *paresthesia* are sometimes met with, under this head including the perverted or abnormal sensations described as numbness, crawling, itching, thrilling, tingling, feelings of weight, of compression or contraction, and of heat and cold. These paresthesias are usually evidences of lighter grades of neural irritation than that causing hyperesthesia.

SYMPTOMATIC FORMS OF TRIGEMINAL NEURALGIA.

Significance of Trigeminal Pain.—In the succeeding section, prosopalgia, or tic douloureux, owing to its great importance, will be discussed separately, this affection, according to the view held by the writer, being due to a chronic sclerotic or degenerative process attacking the Gasserian ganglion. Other important forms of trigeminal neuralgia, diseases with trigeminal pain as their dominating feature, are, however, due to focal lesions affecting the trigeminal apparatus, such lesions, for instance, as tumor, aneurism, abscess, meningitis, exostosis, or periosteal nodes; to neuritis affecting the trunks or termini of the nerves; and to imperfect or perverted supply of blood to the nerve or its centres, the result of disease of the bloodvessels, or of infections or toxins circulating in the blood current. The symptomatic trigeminal neuralgias are sometimes affections of great severity and of wide diffusion so far as the territory of pain is concerned, in some instances involving two or even all of the great sensory divisions of the nerve. A few cases of trigeminal pain are to be referred to neurasthenic and hysterical conditions and to purely functional disturbances.

Irritative Focal Lesions of the Trigeminal Sensory Apparatus.—The effects of focal lesions of the trigeminal apparatus, as of such lesions elsewhere, will vary according as these are irritative or destructive, or both in the same case. Focal irritative lesions cause the symptomatic neuralgias under consideration; and they give rise also to certain trophic disorders. These irritative lesions may be of the Gasserian ganglion, of the peripheral sensory nerves, of the sensory end nuclei in the brain stem, or of the sensory roots.

Trigeminal Neuritis.—Owing to the fact that the head and face are the most exposed portions of the body, neuritis of the trigeminus occurs more frequently than inflammation of nerves in other parts of the body. Causes active in its production are diseases of the ear, teeth, and gums, of the nasofrontal sinuses and of the pharynx. Many cases of head and face pain are due to trigeminal neuritis, although such pain is not infrequently regarded as evidence of meningitis or of intracranial neoplasm. The trigeminus has an extensive distribution within as well as outside the cranium, and especially to the dura. It is quite possible to have a dural neuritis without a pachymeningitis, or even to have a functional disturbance of the dural branches of the fifth nerve without either meningitis or neuritis. When trigeminal neuralgia is symptomatic of a true neuritis, the chief symptom will be pain of greater or less severity according to the degree of the inflammation, and the pain will usually be referred to the nerve distribution affected. Other symptoms will be hyperesthesia over the parts affected with the neuritis, if these can be reached for examination, vasomotor affections such as pallor or

coldness or redness, heat and edema, and trophic phenomena such as herpetic eruptions and changes in the hair. If the disease has continued for some time, the hair may change in color or fall out, as it does sometimes in true tic douloureux. If the affection is chronic and severe, neuroparalytic keratitis, conjunctivitis, corneal ulcerations, and subcutaneous and periosteal thickenings, are other atrophic conditions which may result. Various secretory disorders, such as increase or diminution of the lachrymal, the nasal, or the salivary secretions, may also form a part of the symptomatology. When the cases are of long continuance and great severity, emotional conditions and the evidences of general exhaustion appear. The patient, losing sleep, unable to eat, distressed and wearied by the constant pain, gradually becomes a neurasthenic wreck. Some negative points are important, particularly in distinguishing these forms of trigeminal neuritis from tic douloureux : one of the most important of these is the frequent absence of the paroxysms of facial spasm. Facial spasm may, however, be present in some severe cases of true trigeminal neuralgia (not tic douloureux), especially in those which are caused by intracranial gross lesions. Although both pain and spasm may be severe, they are not usually of the fulgurant character, as in tic douloureux. According to the particular branch of the nerve affected, the symptomatology may acquire special features. Edema of the eyelids with suffusion of the eyes may, for instance, be present in supraorbital neuritis. In some cases when branches of the dental nerve are affected the pain is reflected or radiated to the eye, the ear, or the back of the head. Dilatation of the pupil is an occasional symptom. When the intracranial branches of the fifth nerve are affected, the pain is usually described by the patient as boring or deep-seated, and occasionally it may be referred to one side or one portion of the head, although little dependence can be placed upon such localization of the pain in fixing the exact site of a neuritis within the skull. When vertigo or nausea and vomiting accompany severe and deep-seated pain, the existence of a dural neuritis is not improbable, although the diagnosis cannot certainly be made on this syndrome. Certain painful points or spots are usually found on various portions of the head in the territory of the branch or branches of the nerve affected. The most important of these painful spots for the different forms of ophthalmic neuralgia are (1) the supraorbital point, at or near the supraorbital foramen ; (2) the palpebral, in the upper eyelid ; (3) the nasal, at the point of emergence of the long nasal branch at the junction of the nasal bone with the cartilage ; (4) the ocular, a somewhat indefinite focus within the globe of the eye when the ciliary nerves are affected ; (5) the trochlear, at the inner angle of the orbit. For supramaxillary neuralgia they are (1) the infraorbital, corresponding to the emergence of the nerve from its bony canal ; (2) the malar, on the most promi-

nent part of the malar bone; (3) an indeterminate focus somewhere in the line of the gum of the upper jaw; (4) the superior labial, also indeterminate; (5) the palatine, occasionally the seat of intolerable pain. For inframaxillary neuralgia they are (1) the temporal, a little in front of the ear; (2) the inferior dental, opposite the point of emergence of that nerve; (3) the lingual, on the side of the tongue; (4) the inferior labial. (Ross.)

Trigeminal Neuralgia due to States of the Blood.—Both diffuse trigeminal neuralgia and neuralgias confined to special branches of the nerve are sometimes dependent upon states of the blood,—upon anemias, hyperemias, and especially upon toxemias. Cases of this kind, and particularly for therapeutic reasons, must be differentiated not only from those due to irritative focal lesions, but also from the neuritic forms. It is true that neuritis may originate from a toxemia or perhaps even from a deficient or undue supply of blood to a part, but with our present ideas of the pathology of neuralgias we must recognize the existence of neuralgias of purely hemic origin. In these cases the nerve centres or perhaps both nerve centres and fibres respond with pain because they are starved, overfed, or poisoned, a true inflammation not having taken place. A list of trigeminal neuralgias based upon special causative agents acting upon the blood might be indefinitely extended, but among the most important are those due to anemia, rheumatism, diabetes, gout, lead, malaria, and syphilis. Anemic trigeminal neuralgias occur especially in overworked and badly nourished young women: they may affect any branch of the fifth nerve, but are especially liable to be of the ophthalmic or temporal variety. Rheumatism, gout, diabetes, all may cause a true neuritis, but in some instances they give rise to extreme neuralgic pain without the other symptoms of neuritis, such as hyperesthesia and vasomotor and trophic changes. Lead is a somewhat infrequent cause of trigeminal neuralgias, but oftener attacks the nerves of other portions of the body. Occasionally, however, a striking instance of trigeminal neuralgia due to lead poisoning is observed, and that malaria may be a cause is one of the oldest clinical observations. Malarial neuralgia shows a particular tendency to attack the supraorbital branch of the ophthalmic nerve, causing what is sometimes called brow ague. The great distinguishing feature of trigeminal as of other forms of malarial neuralgia is its periodicity. The attacks tend to return and to augment to a certain intensity at regular intervals, as of one, two, three, or more days, following the same rules in this respect as govern the return of other forms of malarial paroxysms. In speaking of syphilis as a cause of true neuralgia, it must be understood that reference is not made to any of the numerous cases in which this virus causes definite specific lesions like meningitis or gummata, but to those cases in which the action exerted by the poison of the disease in the blood is to disturb

and pervert the normal functions of the nerve centres. Such cases, while perhaps not rare, are difficult of exact diagnosis, and with a history of syphilis we are likely to fix our attention upon the probability of a localized lesion rather than upon the possible effects of a toxic agent in the circulation. Trousseau, and others following him, have described a form of *epileptiform trigeminal neuralgia* which is regarded by some as distinct from prosopalgia, or tic douloureux, and by others as a variety of this affection. We incline to the latter opinion. In this disease the patient may have daily one or many attacks of lightninglike pain, which succeed one another with great rapidity for a few seconds or minutes and then disappear. The attacks of pain may recur for days or weeks and then may disappear for months, or even for a longer period, but, as a rule, the disappearance is not permanent.

Diagnosis, Prognosis, and Treatment of Symptomatic Trigeminal Neuralgias.—The etiology and pathology of the symptomatic trigeminal neuralgias have been sufficiently indicated in the description of these affections, which in our consideration of them have been largely classified from the etiological and pathogenetic standpoint. Their diagnosis is largely dependent upon a close study of their history and causation. In the first place, the different classes of neuralgia of this type must be carefully separated from one another: for instance, those due to focal lesions from the neuritic and toxic varieties. Safe therapeutic tests will sometimes be of great service in clinching the diagnosis. It may be found that the cases yield primarily to an antirheumatic, antilithic, or antisyphilitic treatment. The urine and blood should be examined, in doubtful cases, to determine the presence or absence of diabetes and special blood states. Diabetic neuralgias are said to show a tendency to bilateral symmetry, to attack the same portion of the nerve distribution on both sides. Neuralgic affections must be carefully differentiated from the pains which indicate intracranial lesions, and especially from tumors in their initial stages. The prognosis of symptomatic trigeminal neuralgias is comparatively good, but depends upon the particular variety. Many of the constitutional forms are amenable to treatment. Those due to focal lesions may or may not be, according to the extent and the character of the lesion. Neuritis is especially intractable when it attacks the intracranial branches of the fifth nerve, but even in these cases it may sometimes be reached by treatment. The epileptiform neuralgia of Trousseau is practically incurable. The most that can be said of it is that the patient may, by good fortune, remain free from the attacks for a longer or shorter period. In the treatment of symptomatic neuralgias the matter of first importance is to seek and attack the cause. Anemic neuralgias are best treated by fresh air, an abundance of good food, and the use of iron, arsenic, and manganese. Mercury and

the iodides are the therapeutic anchors in syphilis, the salicylates in the rheumatic form, and lithium and colchicum in the gouty form. In diabetic neuralgias the remedies of most value are the salicylates and the preparations of codeine. One of the chief indications must always be the immediate relief of the pain. As the measures to accomplish this object are practically the same in the symptomatic neuralgias as for tic douloureux and migraine, the treatment as given under these affections can be consulted.

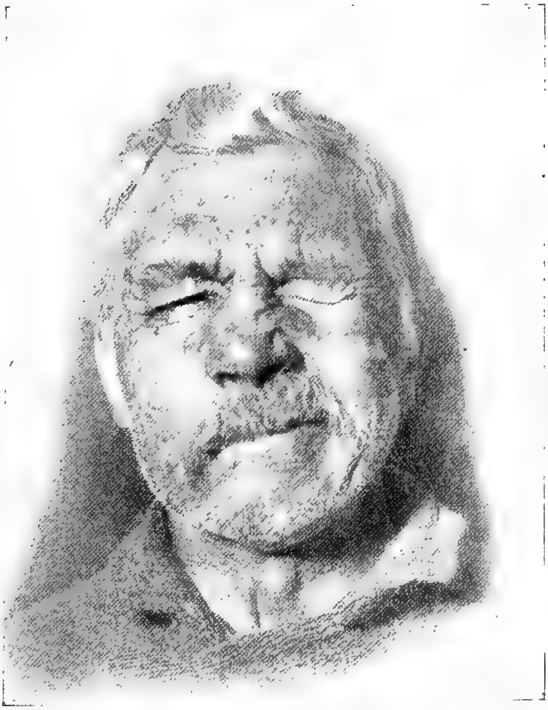
PROSOPALGIA, OR TIC DOULOUREUX.

Definition and Synonyms.—Prosopalgia is a form of trigeminal neuralgia of extreme severity, paroxysmal in type, and usually having associated with the pain attacks of spasm involving the muscles of the face. We have used the term prosopalgia as best because, meaning simply "pain in the face," it does not involve any theory as to the local or other cause of the disorder. It should, however, be regarded as a severe type of trigeminal neuralgia. Its most common synonyms are *tic douloureux*, *trifacial neuralgia*, *facial neuralgia* (not a good name, because misleading as to the nerve affected), and *Fothergill's face ache* or *neuralgia*. This form of trigeminal neuralgia is a special disease, and should be clearly separated from those forms of trigeminal neuralgia which depend upon inflammatory or other disease of special portions of the nerve. These have been considered in the preceding section of this chapter.

Clinical History.—In typical cases the disease presents a somewhat uniform method of onset, development, and climax. The patient, frequently in or past middle life, is without warning attacked with sharp pains in some special portion of the face, as in the upper or the lower lip, near the angle of the jaw, under the eye, or on the forehead. These pains, ordinarily intense and lancinating, may soon pass away, but they generally recur, and as the case progresses usually become more severe and diffuse. Often they remain for a long time in the portion of the distribution of the nerve first attacked; later, pain appears in other branches of the same subdivision, or it may appear in an area of another of the divisions. Beginning in one of the subdivisions of the third branch, eventually the third, second, and first may all be more or less implicated in the painful attacks. The second and first and the third and second are more likely to be conjointly involved than the third and first. This tendency to diffusion from the subdivisions of one of the three great divisions of the nerve to subdivisions of one or of both of the other branches throws light upon the probable nature of the disease, to a certain extent favoring the view of its ganglionic origin. As the disease progresses, various exciting causes will readily bring on the attack of pain. The paroxysm may be precipitated, for instance, by drinking, by touching the face, by moving the jaws, by the slamming of a door,

by the noise of the falling of an object, or by anything tending to cause actual movement or to produce emotional disturbances. The patient lives in a state of constant anxiety, fearful of everything. The patient's face may become hyperemic or even suffused and swollen, or the conjunctiva alone may be injected. Excessive lachrymation may be present. In some cases unilateral sweating, and in rare instances trophic changes, such as attacks of herpes and discoloration of the skin and possibly thickening of the skin, may be observed. Hearing, taste, and smell are rarely disturbed, but vision is oftener affected. The usual disturbances of vision are transitory paresis of the ocular muscles or trophic changes in the retina. Spasm in the facial muscles accompanies the paroxysms of pain, and this spasm is often very violent, and has given the affection one of its names, *tic douloureux*. In the illustration (Fig. 410) this spasm is well shown, the face being forcibly drawn to the side affected. The ocular muscles are sometimes spasmodically involved, as are also the muscles supplied by the motor subdivision of the fifth nerve. Occasionally the patients become hallucinatory or even maniacal. Féré reports a case in an epileptic complicated by zona in which menacing voices were heard on the affected side, but the patient was conscious that the voices were unreal. Intense sialorrhea came on with the neurotic attack, and the voices disappeared with them. It is probable that in this case the epilepsy predisposed to the hallucinations. As a rule, tic douloureux is more severe and recurs with greater frequency in winter and spring.

FIG. 410.



Case of tic douloureux: appearance of the patient's face during one of the severe paroxysms.

Etiology.—Prosopalgia is more frequent among women than among men. It occurs usually in or after middle life, and is a not

infrequent disease of the aged. It is very rare in childhood and youth. The tendency to its occurrence is marked in neuropathic families, and especially in families in which epilepsy is present. Toxic and infectious agents probably act as exciting causes ; certainly anything which tends to lower the tone and vitality of the system will lead to tic douloureux in those who have a constitutional tendency to the affection. It will be remembered that Anstie held that neuralgia was the prayer of the starved nerve for blood. In this consideration of the etiology of tic douloureux I have purposely omitted the discussion of the numerous focal lesions which are usually enumerated as causative factors in its production, believing that the disease is essentially an irritative degenerative affection of the Gasserian ganglion, nearly all its so-called causes being simply exciting factors in those in whom a persisting pathological state predisposes to the disease.

Pathogenesis.—The history of the development of the disease, the paroxysmal nature of many of its symptoms, its associated phenomena, and the unsatisfactory results of treatment lead to the conclusion that the true seat of prosopalgia, or tic douloureux, is in the Gasserian ganglion. Although pathological proof is still wanting, the disease is in all probability a form of degeneration of the cells of this ganglion, similar to that which occurs in the dorsal spinal ganglia in tabes, and to the degeneration of the ganglion cells of the anterior horns in chronic atrophic affections, such as amyotrophic lateral sclerosis and progressive muscular atrophy. It is probable that in sensory cases the disease gradually invades all portions of the ganglion, and as one cell after another drops out the instability of the ganglion is increased, and with this the tendency to violent sensory discharges. This theory seems to me more probable than that which attributes the affection to a thalamic or cortical origin. As the central sensory neurons have their cells of origin in the Gasserian ganglion, and possibly in the thalamus, the discharging lesions which give rise to the violent paroxysms of pain have their true seat and source in one or the other of these cell masses, but most probably in the former.

Pathological Anatomy.—In some cases forms of low grade neuritis have been discovered in branches of the nerve affected. The vessels which supply the nerve with blood are sometimes affected with chronic inflammatory processes, but in a large number of cases branches of the nerve which have been removed by operation have been examined without discovering any noteworthy alterations in the nerve. In three cases in which Dana examined four superior maxillary nerves, striking evidences of arterial disease were found, and in a fourth case no bloodvessel was present in the specimen. This led him to the view that an obliterating arteritis was a factor in the disease.

Diagnosis.—The diagnosis of prosopalgia is usually readily made. The only point of importance is to distinguish between that form which has been considered in the present section, which is due to a chronic degenerative process of the Gasserian ganglion, and the forms which are due to focal irritative lesions, such as neuritis, exostosis, cicatrices, tumors, meningitis, etc., located somewhere in the course of the trigeminal sensory apparatus.

Prognosis.—The prognosis of prosopalgia is almost invariably bad. Periods of relief from suffering are obtained either by treatment or without reference to treatment. Unfortunately in the vast majority of cases the disease gradually grows worse, both as to the frequency with which paroxysms occur and as to their severity. On the whole, those patients do best who early submit to operation, probably because even the temporary relief which they obtain—a relief which often extends over months, or it may be years—allows time for recuperation.

Treatment.—A large number of medicinal remedies have been used for the relief of prosopalgia. Those which appear to afford most relief can be classified under the two heads of constructives and anodyne sedatives. Nearly all sufferers from prosopalgia receive some benefit from rest and the use of tonic and building-up remedies like cod liver oil, preparations of iron, arsenic, manganese, and the metallic tonics generally. Strychnine sulphate in increasing doses has proved of great benefit when the treatment has been continued over a number of weeks or months, and nitroglycerin is of particular value in the aged or prematurely senile, acting through its well known effect upon the vessels. Preparations of aconite, and especially the alkaloid aconitine, are also useful both in relieving the pain and in changing the character of the disease, and among other remedies for the relief of the pain are morphine, codeine, phenacetin, antipyrin, antifebrin, and ammonol. Morphine or codeine, if given in sufficient doses, will usually give relief, but opium preparations should not be given too freely, as the patient may acquire a drug habit. Hot whiskey and quinine will sometimes temper the violence of an attack. All constitutional conditions, like anemia, rheumatism, gout, diabetes, and syphilis, which predispose to the neuralgic attacks should receive attention, and should be treated with the drugs which are known to be beneficial in these affections. In special instances the salicylic preparations will prove of great value, and the best of these are the sodium salicylates and oil of gaultheria. From ten to fifteen grains of the former, or from ten to twenty drops of the latter, should be given three or four times daily. When, as is too frequently the case, tic douloureux does not yield to other treatment and continues to render the life of the patient almost unendurable, resort should be had to surgical treatment. Even when a permanent cure is not obtained, the few months or few years of relief afforded

enable the patient's general health to be built up, and give him the opportunity of enjoying life during the period of respite. The most important surgical measures are stretching, resection, or avulsion of the extracranial portions of the nerve, section of the nerve trunks in front of the Gasserian ganglion, and operation on this ganglion. For the various methods of operation surgical works should be consulted. The choice of operation must depend to some extent on the severity but especially on the diffusion of the pain. When this is confined to a single accessible portion of the nerve, neurectomy should be tried, or neurectomy and avulsion. When the neuralgia affects two or more of the great divisions of the nerve, the Gasserian or pre-Gasserian operation deserves full consideration, although even in some of these cases separate operations on different nerve trunks seem to afford the most relief. In the light of what has been said of the pathology of prosopalgia, it may be thought that no operation except that on the ganglion has a rational basis; but this does not seem to me to be the correct view to take. While the disease is probably a degenerative and irritative process, attacking the cell bodies of this ganglion, it must be remembered that the nerve trunks themselves are composed of the peripherally distributed axis cylinder processes of these cells, and that their section may reflexly inhibit the discharging process which causes the pain or may prevent the transmission of the painful impulses. In many cases experience has taught that neurectomy and avulsion afford relief for a considerable period, but, on the whole, the extirpation of the Gasserian ganglion has afforded the most permanent relief.

MIGRAINE.

Definition, Synonyms, and Varieties.—Migraine is a periodical paroxysmal affection, usually hereditary, and chiefly characterized by severe attacks of pain confined to one side or to one portion of one side of the head, and commonly associated with either nausea or vomiting or both. The synonyms for migraine are *megrim*, *sick headache*, *neuralgic sick headache*, *paroxysmal headache*, and *hemicrania*. Migraine in the first place presents itself in what might be called its most typical form, in which all the phenomena are of great severity, the pain extending over a large part of one side of the face and head, and in some cases radiating to all parts of the head, although the focus of greatest severity is usually in the temporal or the supraorbital region of one side. Special varieties are determined chiefly by the limitations of the painful areas; but the only one of these that will be separately considered is the migrainoid supraorbital neuralgia.

Symptomatology.—*Miscellaneous Symptoms.* Not infrequently the patient has decided prodromes, usually in the form of general malaise, discomfort, or depression, which may last for hours or days.

Photophobia, visual spectra, tinnitus, vertigo, and gastric disorder may be other prodromal signs. The attacks frequently show a great tendency to periodicity, a fact to which special attention has been called by Liveing and Sinkler. The latter has reported a number of instances indicating the tendency of the disease to recur on fixed days. Salius relates the case of an Italian monk who for three years and seven months had an attack of violent hemicrania every Monday, the attacks lasting from twenty-eight to thirty hours (Tissot). Usually beginning in one side of the head, the pain augments in violence, sometimes slowly, sometimes rapidly, until finally the patient is compelled to take a recumbent position. It is often of a throbbing character, the patient complaining that the head feels as if it would burst. It is increased by noise, light, jarring movements, and anything which causes emotional excitement. The appearance of the patient differs in different cases. Frequently the face is pale; both the head and the limbs feel cold to the patient. In other instances the opposite appearance may be present, the face being flushed or even turgid, and in still other cases alternations of pallor and of flushing or turgescence may occur. In rare cases one side of the head and face may be pallid and the other flushed. These differences in vascularity and in temperature are among the clinical reasons which have led to the belief that the disease should be subdivided into an angiospastic and an angioparalytic variety. The pupils may be either contracted or dilated, myosis usually accompanying pallor of the face, and mydriasis flushing. Forms of oculomotor paresis are comparatively common, and have already been referred to when discussing affections of the third nerve, page 844. Some would have us regard the cases of migraine with these oculomotor symptoms as a special type, which has been designated *ophthalmoplegic migraine*, but no reason for such classification exists greater than might exist for describing an auditory form or other forms based upon the frequent presence of certain special symptoms. The mental condition of the patient varies somewhat with temperament. Frequently great apprehension is felt, and nearly always a feeling of depression. The ideas may become confused, and the patient may have hallucinations of sight or hearing. Nausea and vomiting are frequent accompaniments of the headache, and have given it one of its commonest names, sick headache. Sickness of stomach comes on early in some, in others not until the paroxysm has persisted for a number of hours. An attack of copious vomiting, either spontaneous or brought on by the use of emetics, often marks the climax of the attack, the patient afterwards sinking into a sleep, and the pain gradually disappearing. The pulse is usually small and tense, and is more likely to be lessened than to be increased in frequency. The duration of an attack is from eight to twenty-four hours, although it may last two or more days. While the affection, as has

been stated, exhibits a considerable tendency to periodicity, in many cases the paroxysms recur somewhat irregularly, varying from one week to two or three months. In children the symptoms are somewhat less pronounced.

Visual Spectra (*Ophthalmic Migraine*). Reference has already been made in several places in preceding sections to the spectra of migraine and the forms of fugacious hemianopsia with phosphenes and other visual phenomena sometimes present preceding or during an attack of migraine (pages 757 and 773). It has been stated that the central field is nearly always free from visual spectra, and also that various disturbances of the mechanism of cerebral speech occur. The disturbances of vision are of such dominant importance in some instances that it has been suggested that *ophthalmic migraine* should be regarded as a special form of this disease (Charcot). This so-called ophthalmic migraine may present itself in a simple form in which only headache and visual phenomena are present: Gowers refers to one curious case in which visual disturbances exactly such as precede attacks of migraine occurred frequently during many years as an isolated symptom, but at no time was there any headache or pain. In other cases various symptoms of cerebral disturbance are combined with the headache, and visual spectra. The patient, for instance, may suffer from transient and incomplete aphasia, and this may or may not be associated with hemiparesis or monoparesis or with paresthesia like numbness and tingling confined to one side of the body or of the face or to the limbs of one side. In rare instances transient aphasia is the only evidence of an attack which is fundamentally the same as that of migraine, a fact which should be remembered, as the absence of headache and of the other phenomena of the disorder may lead to the belief that the patient is suffering from a real but limited apoplexy. Charcot has directed attention to the occurrence of ophthalmic migraine as a prodrome of general paralysis of the insane. In other rare instances migraine does not set in until after the disease has been initiated as indicated by other physical or mental symptoms. In most cases the migraine cannot be regarded as having any direct connection with the general paralysis. It is a concomitant and forerunner only in the sense that among the lesions productive of the disorder happen to be some which are so situated and of such character as to give rise to ophthalmic phenomena. Much space could be filled with a description of special types of visual phenomena recorded by different authorities as occurring in migraine. Gowers in particular has elaborately discussed this subject. Among such recorded visual spectra are the zigzag "fortification lines;" luminous objects, or objects encircled by luminous rings or fringes of the most brilliant colors; visions of moving animals, sometimes pleasant and sometimes as disagreeable as those of delirium tremens; and dark and bright spots, cometlike and

lightninglike flashes, and balls of fire which sometimes dissipate into scintillating sparks or flakes.

Migrainoid Supraorbital Neuralgia. The question has arisen as to the relation of some of the forms of supraorbital neuralgia to migraine. Putnam has particularly studied these cases, which, according to him, arrange themselves into several groups: (1) those in which no migrainoid or other special neuropathic tendency is traceable; (2) those in which other members of the family have had this same form of migrainoid neuralgia, with perhaps a touch of true migraine; and (3) those in which the patient's attacks and those seen in other members of the family approach very nearly to the true migraine type. He gives the history of cases illustrative of these different varieties. The affection in question could perhaps, therefore, be properly treated of either under migraine or under symptomatic trigeminal neuralgia.

Etiology.—Heredity is by far the most important predisposing cause of migraine. I have been able to trace its occurrence through five generations. In many inherited cases it begins very early in life. According to Gowers, one third of all the cases begin between the ages of five and ten years. In a personal case with a clear history of heredity extending through several generations, the child was first attacked with migraine between the ages of two and three years. Even in the majority of cases which begin after youth or manhood has been reached, an hereditary element can be found on search. Often when the existence of migraine cannot be discovered in the direct ancestry of the patient he has inherited a neuropathic constitution, as indicated by the occurrence in his forebears of epilepsy, insanity, hysteria, alcoholism, tabes, or of some other form of degenerative nervous disease. General debility, and the depressing effects of infectious diseases or toxic agents, may act as predisposing causes. In the study of the etiology of migraine, two mistakes are sometimes made: one, in confounding other forms of headache with migraine, and the other, in giving undue weight to causes which are merely exciting. The causes of symptomatic trigeminal neuralgia or of headache of trigeminal origin in cases which are distinctly not of the migrainoid type are sometimes assigned as the causes of true migraine. This remark applies to not a few of the forms of headache and trigeminal neuralgia which are of toxemic origin and which have been already considered; nevertheless, it is true that a rheumatic, gouty, saturnine, syphilitic, or other abnormal state of the blood may precipitate attacks of migraine in those who are hereditarily predisposed. Those forms of symptomatic headache which are solely due to nasal, pharyngeal, and aural diseases are certainly in the vast majority of cases not of the migrainoid type, and the same is true of most of the headaches which have been attributed to carious teeth. The relationship of eyestrain and the

effects of the correction of refraction errors, of tenotomy, and of visual hygiene in the relief of headaches is of interest in this connection. The headaches which are caused by the visual and ocular defects and which are relieved by the above measures may or may not be migraine, and it is misleading to make sweeping assertions as to the causation of migraine by such abnormalities. The truth with regard to all these so-called causes of migraine is simply that individuals who are predisposed to this disease, and who have had attacks of it of greater or less severity, are liable to have paroxysms of migraine excited and the frequency of their recurrence augmented by such causes as abnormal states of the blood, nasal, aural, and pharyngeal lesions, dental irritation, and ocular and visual defects. Intellectual capacity has been thought by some to play a part in the etiology of migraine. Certainly many men of great distinction and unusual mental powers have been affected by this disease; but it may occur in any walk of life, and the two most reasonable explanations of its occurrence in the higher classes intellectually are hereditary predisposition and the commonly better habits as to exercise and fresh air of those who follow nonintellectual occupations. Migraine is more common in women than in men in the proportion of three or four to one. The attacks occur with most frequency in winter and early spring.

Pathology.—The pathology of migraine still remains unsettled, good authorities taking views widely diverse or apparently so, for in some instances the differing views seem simply to be different methods of stating the same opinions. Migraine may be regarded as (1) a form of trigeminal neuralgia of Gasserian or neural origin; (2) a discharging neurosis either of thalamic or of cortical centres; or (3) a vasomotor neurosis. Anstie believed that it was a neuralgia of the ophthalmic division of the trigeminus due to processes of degeneration going on in the nucleus of origin of the nerve, a theory which would bring migraine into close relation with tic douloureux according to the pathology of the latter affection adopted by the writer. Migraine, however, while presenting some of the features both of tic douloureux and of the symptomatic neuralgias, has others which belong to neither of these affections, and its pathology must therefore, in part at least, be different. The pain of migraine is located in the distribution of the trigeminus, but in true migraine the trigeminal apparatus is disturbed throughout,—from the Gasserian ganglion to the sensory cutaneous termini in one direction, and in the other to the end nuclei, root bundles, and cortical termini. Even if the affection in its manifestations is in part to be classed as a vasomotor neurosis, the angiospasm and angioparalysis are phenomena associated with those of trigeminal irritation. With Moebius, Putnam, and others, we must agree that it is a disease which is the outcome and sign of hereditary degeneration. It is probable that the

underlying weakness which leads to its manifestations is distributed in all portions of the trigeminal apparatus and to that part of the gangliated nervous system which is associated in action with this apparatus. With Putnam, I believe that the study of migraine, like that of other serious painful affections, should be based on broader and deeper investigation of the physiological functions which the sensory nerves subserve; that we have in these disorders "a sort of caricature of physiological processes, or bits of these processes cut off from their normal relations."

Diagnosis and Prognosis.—Migraine is a disease the symptomatology of which is so clearly defined that in most cases its diagnosis can be readily made. It is perhaps most likely to be confounded with headache of organic origin, particularly with that due to tumor or pachymeningitis. The diagnosis in this case can be made by close study of the various localizing and general symptoms of tumor or meningitis, and will be much aided by the use of the ophthalmoscope and the history of the case. Those forms of migraine which give visual spectra or varieties of aphasia with head pain can be diagnosticated only by the history of other attacks of migraine or by the paroxysmal and transient character of the affection. Migraine and tic douloureux are not likely to be confounded, except in the case of imperfect and irregular types of either. Hysterical or imitative headaches in children may occasionally closely simulate migraine, particularly in children whose parents are victims of this disease. A diagnostic difficulty, to which Gowers has especially called attention, is that presented when the subjects of migraine become affected with some other disease, the symptoms of the two affections then complicating one another in a confusing manner. Among the most important of these complicating disorders are Bright's disease, brain tumor, and meningitis. So far as absolute recovery is concerned, the prognosis of migraine is bad. In most cases it shows some tendency to improvement when middle life is reached, the attacks becoming less frequent and less severe.

Treatment.—*Hygienic Measures.* While migraine is essentially an incurable affection, one that depends upon inheritance and the predisposition to which disappears only with the natural changes wrought in the individual, much can be done not only to palliate the paroxysms of the disease, but also to render them less frequent and to reduce their power to cause suffering. No one line of treatment seems to be applicable to all cases, and yet certain therapeutic measures are of advantage in almost any case. First should be considered the means to be taken to counteract the inherited predisposition, to cure the individual approximately of the tendency to the attacks. The most important measures are those which are calculated to maintain the nervous and general health of the patient at the highest possible level. Close attention should be paid to the

general hygiene and diet of predisposed children, as improper food, undue application to school studies, and sedentary habits excite attacks of migraine. The roborant treatment applicable to adults may be of an opposite character in different cases. Seclusion, rest, and full feeding may, for example, be of particular value in some cases, while fresh air, abundance of exercise, and free association with others may be of equal service in those of different temperament and habits. Climate plays a part of great importance in the prevention of migraine, although as yet no clear data with regard to the details of climatic treatment are forthcoming. Sinkler speaks of a young lady under his care who suffered from migraine of the severest type, and in whose case no plan of treatment or regimen seemed to have any influence upon the attacks, but going to the far West for some months she remained free from the attacks during the entire time she was there. Outdoor life as well as change of climate plays a part in some of the cases in which the benefit is attributed solely to the latter. One of my friends, who has inherited migraine and who suffers at times from severe attacks, remained entirely free from them while camping out in the far West, where for a long time he spent several months in each year in the service of the government. During sixteen or seventeen years he never had an attack while in camp. With regard both to this case and to the one referred to by Sinkler it is to be borne in mind that the immunity which they had from the attacks was during the summer months, but in both of these cases also the individuals suffered at times from the migraine in summer, as in other seasons, when not in the West. The forms of outdoor exercise now so popular are distinctly beneficial to the subjects of migraine. In a number of instances I have known patients who have suffered for years to have fewer attacks in periods of the same length after taking up bicycling, which, if indulged in with moderation and good sense, is highly beneficial in nervous affections as well as in migraine. Sea bathing is of service, especially if the subjects of migraine attend also to diet and exercise.

Treatment of the Attack. Among the drugs which have in recent years attracted the most attention in the treatment of the paroxysms of migraine are phenacetin, antipyrin, antefebrin, caffeine, sodium salicylate, and ammonol.* The reports as to the use of phenacetin are conflicting, but are favorable for the majority of cases. Doses of from five to ten grains are most commonly used, but doses as large as a drachm or more have been successfully employed. It is not, however, so generally useful as some of the other drugs deservedly popular in the treatment of migraine. Antipyrin was first used in the

* An excellent *résumé* of the medicinal treatment of migraine is given by Dr. Wharton Sinkler in a paper read before the Association of American Physicians and published in the *Medical News*, July 18, 1890.

treatment of migraine, or at least of headache, by White. Sinkler has collected the statistics of its use in a large number of cases, in the vast majority of which its action was satisfactory. Some of its advocates are in favor of large doses, as from ten to fifteen or twenty grains; others favor smaller doses, as three or four grains. My own experience is in favor of the administration of from three to six grains at comparatively short intervals. Antifebrin is, on the whole, of more value than antipyrin, and probably is somewhat less dangerous, and should be used in doses of from ten grains to half a drachm. One of the most valuable remedies to relieve or mitigate the attacks of migraine is caffeine, which may be given either in the form of the alkaloid or as caffeine citrate. Guarana, of which caffeine is the active principle, seems in some cases to be of more service than the caffeine itself, as evidenced by the popularity of some of the guarana mixtures. A chemical mixture of antipyrin, citric acid, and caffeine is an efficient preparation in relieving the severity of the paroxysms of migraine; it is sold under the name of migrainin. Another really efficient remedy, but one whose repeated use is liable to lead to abuse, is bromocaffeine. With some patients it acts as a mild cathartic as well as a sedative, and its value is probably enhanced in this way. In some instances I have found ammonol the most efficient and least depressing of the newer remedies for the relief of migraine and some of the forms of symptomatic neuralgia. It can be used effectively in doses of from five to twenty grains, and one of the best methods of dispensing it is in capsules. Amyl nitrite and nitroglycerin have both been recommended on physiological grounds, but if useful are of benefit in only a limited number of cases, probably in the so-called angiospastic variety. Among remedies which assist in allaying nervous excitement are the bromides, chloral, croton chloral, chloralamid, trional, and sulphonal. Lithium bromide is more efficient in equal dose than the other bromides. Fifteen or twenty grains of lithium bromide with or without five grains of chloral will sometimes abort a threatened attack by quieting the nervous system and inducing sleep. When prodromes habitually precede the fully developed attack for a considerable period, the use of a saline cathartic and seclusion in a quiet room may also abort an attack. When the attacks come on after eating, or when the process of digestion has been arrested, as it sometimes is when an attack of migraine is foreshadowed, no measure is so efficient as free emesis, which can be induced by draughts of hot water or hot mustard water, or by the administration of ipecacuanha, or by the hypodermatic injection of apomorphine. Much relief is afforded during the attacks by the local measures which are so commonly employed, such as firm pressure on the head, the application of towels dipped in hot water or of hot water bags, and stimulating applications like mustard plasters to the nape of the neck. With some patients cold

to the head is more efficient than heat, and this is usually best applied by repeatedly dipping towels into ice water, or by the use of the ice bag. At the same time that the very hot or very cold water is applied to the head the feet may be bathed in a hot mustard bath. All forms of electricity have been used in the treatment of migraine, both for the paroxysm and in the intervals between the attacks, and weak galvanic currents carefully applied to the head certainly sometimes afford relief. One electrode can be placed on each mastoid process, or one on the back of the neck and the other on the forehead. The current should be passed for one or two minutes. Mild, rapidly interrupted faradic currents applied by the hand of the operator to the patient's head also at times afford some relief. With regard to the migrainoid intermittent headache, this variety of the disease is far more amenable to treatment by quinine than is the ordinary type of migraine. The quinine needs to be given in large doses, from fifteen to thirty grains daily, and the best plan seems to be to give the whole quantity in one or two doses about four hours before the attack is due. (Putnam.) The local treatment of the nasal passages is also of great importance, as this treatment may indirectly improve the condition of the frontal sinuses.

Treatment in the Intervals between the Attacks. Among medicinal remedies which have been recommended by a large number of those who have written on the subject of migraine, or who have had much practical experience in the treatment of the disease, cannabis indica holds a high place. This drug should be given during the intervals between the attacks, as it seems to have some influence on the disease itself if its use is long continued. James Little recommends for the intervals between the attacks a pill of sodium arsenate, extract of cannabis indica, and extract of belladonna, of each one half grain. In addition to this pill, two grains of valerianate of zinc given twice daily, and, to cut short the paroxysms, twenty grains of sodium salicylate in a wineglassful of water, made effervescent by a dessertspoonful of granular citrate of caffeine, should be administered. Another prescription which may be used with advantage, composed of cannabis indica, nux vomica, and ergotine, is given on page 246. Some patients are intolerant of cannabis indica, and in others if care is not taken the cannabis habit may develop. When migraine is associated with the gouty diathesis the lithium salts and natural mineral waters should be freely employed. Haig states that when the uric acid diathesis is present he has relieved many attacks by giving from twenty to thirty drops of dilute nitrohydrochloric acid, well diluted, and repeated once or twice at intervals of half an hour. In the discussion of etiology my views with regard to the relations of eyestrain to migraine were stated. The favorable results of ocular treatment have undoubtedly been overestimated, yet measures directed to the eyes should not be neglected. The various disorders

of refraction should be corrected if sufficient to cause discomfort or annoyance, but the hope should not be held out of a radical cure of the migraine, although the evidence seems to be fairly conclusive that paroxysms of migraine are less frequent and of less severity after the relief of eyestrain. Tenotomy and partial tenotomy should be performed only when the indications are for their performance independently of the existence of migraine.

TRIGEMINAL REFLEX NEUROSES.

Trigeminal Headaches.—Diseases of the trigeminus might be greatly extended if we would accept the idea of classifying under them all the affections which have been referred to irritation of peripheral filaments of this nerve, or to one or another sort of toxic action exerted upon the nerve or its centres. Not a little of modern rhinological and laryngological practice is founded upon the idea that various disorders, cephalic, ocular, respiratory, cardiac, and general, are dependent upon irritation localized to terminal filaments of the trigeminus. Among the affections which are most frequently discussed from this point of view are certain varieties of headache. Headache in one sense may be said to be almost always of trigeminal origin, as the trigeminus is the main sensory nerve of the head. Even if pain is directly due to disease or disturbance of the sensory regions of the cerebrum, these in part constitute a portion of the trigeminal apparatus. As has been shown in various places, the pain of brain tumor, meningitis, or abscess, and of other focal and diffuse encephalic lesions, is due directly or indirectly to trigeminal irritation. It is sometimes important to distinguish between headaches or head pains due to direct irritation of the dura, and those which seem to be dural, or at least intracranial, but are really due to irritation reflected from points outside of the skull, as this distinction may enable us to differentiate between a serious intracranial affection and one more amenable to treatment because due to a lesion of an accessible region like the nose, the ear, or the throat. According to Allen, the reflex headache is almost entirely restricted to the temple, the forehead, and the vertex, and this author even locates the nasal disorder with reference to the head pain. He has found in all varieties of chronic catarrh that a dull pain in the region of the forehead has been complained of, and when the disease is confined to that portion of the nasal chamber which corresponds to the middle turbinated bone the pain is referred to the temple; beginning in the forehead and temple, in some cases it extends to the vertex and the nape of the neck. It may be accompanied with nausea and vomiting, and then be regarded as a true headache or attack of migraine. Allen distinguishes reflex catarrhal headache from sick headache of gastric origin by the absence of gastric disturbance; from the temporal pains of eyestrain by its persistence after the correction of the

errors of refraction; and, with less exactness, from neuralgia of the head by exclusion of the rather multiform causes which lead to this condition. While the headaches to which Allen refers are doubtless of nasal origin, it might be questioned whether they should be regarded as reflex headaches. It might be more correct to speak of them as headaches due to direct irritation of the terminal filaments of the trigeminus, they being reflex only in the sense that the patient refers them to an intracranial position although the cause is extracranial.

Trigeminal Reflex Cough.—Under the name of *trigeminal cough* Hirt calls attention to a reflex neurosis which has also claimed the attention of others who attribute the cough entirely to irritation of the trigeminal fibres distributed to the nose, pharynx, or external auditory meatus. Subcortical varieties of the reflex neuroses, according to the site of the initiating lesion, have been described, as nasal, pharyngeal, and auricular. It is by no means a rare affection. Among the best methods of treatment are cauterization, the nasal douche, and the use of weak faradic currents to the nasal cavity or to the throat. Besides cough, forms of paroxysmal asthma and of vertigo have been assigned to trigeminal irritation, as have also several forms of vertigo. Certain facts with regard to the effects that can be produced by irritation of the trigeminal filaments in the nose or the conjunctival sac favor the view of the trigeminal reflex source of certain cases of cough, asthma, and vertigo. It has been found, for instance, that irritation of the trigeminal nasal filaments or conjunctival filaments cuts short laryngeal spasm.

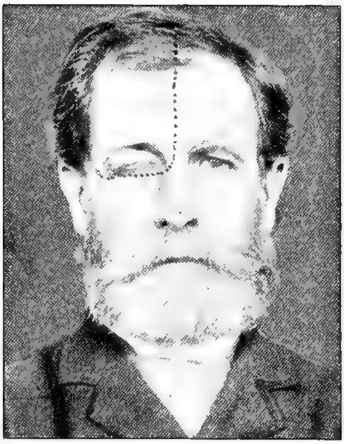
ANESTHESIA FROM DISEASE OF THE TRIGEMINUS.

Trigeminal Anesthesia due to Focal Lesions.—*Positions of the Lesions.* Anesthesia more or less complete results from a destructive lesion of any portion of the trigeminal apparatus,—from lesion of the Gasserian ganglion, of the peripheral branches of the sensory portions of the nerve, of the sensory root between the Gasserian ganglion and the pons, of the entering root fibres in the pons, of the sensory end nucleus in the oblongata, of the descending spinal root, of the descending sensory root, of the central sensory pathway in the peduncle and the internal capsule, of the thalamus, and of the sensory cortex and subcortex. A glance at the diagram of the trigeminal apparatus (Fig. 402) will show the positions in which the lesions just enumerated might be located. Lesions causing pressure on any of these parts will produce transient anesthesia, which may become in part permanent if the pressure interferes with the nutrition and leads to degeneration.

Anesthesia from Disease of the Trigeminal Nerve Trunks at the Base of the Brain. More or less limited or diffuse anesthesia may be produced by a tumor at the base of the brain, a basal meningitis or

abscess, a cavernous sinus thrombosis, a peripheral neuritis of rheumatic, syphilitic, or other origin, fractures of the floor of the skull, traumatisms cutting, crushing, or bruising branches of the nerve after their exits from the cranial foramina, or toxic agents acting upon any portion of the nerve from its centres to its termini. In many cases, as has already been sufficiently indicated, disturbances of the motor portion of the trigeminus or of other adjacent and correlated structures may be associated with the anesthesia. A somewhat frequent association is anesthesia of the ophthalmic branch of the fifth nerve, with total or almost total paralysis of the third. In Fig. 411 is an illustration of such a case reported by McConnell from the Polyclinic service of the writer. This patient had a history of syphilis, although his paralysis apparently came on as an immediate result of exposure to a draught, after which he was seized with sharp pain in the eyeball and the right frontal region. This was followed by an herpetic eruption, and still later by burning sensations, and soon the right upper eyelid was paralyzed. Anesthesia was complete over the right forehead, temporoparietal region, and eyelids, and the cornea was cloudy and insensible to touch, but not ulcerated. All the extraocular muscles supplied by the third nerve were paralyzed, as were also all the intraocular muscles, both cycloplegia and iridoplegia being present. In this case an inflammation syphilitic or rheumatic in character probably affected the sheaths and trunks of both the third and fifth nerves. Schlesinger has reported a case with complete paralysis of the oculomotor nerve of the left side with numerous pigmented spots on the forehead the result of herpes zoster, the paralysis having occurred one week after the herpes.

FIG. 411.



Paralysis of the third nerve, and anesthesia in the distribution of the frontal and lachrymal branches of the fifth nerve. The area of anesthesia is within the dotted line.

Trigeminal Anesthesia from Pontile and Oblongatal Lesions. Gross pontile lesions causing trigeminal anesthesia frequently also involve the sixth, seventh, eighth, and ninth cranial nerves. The forms of trigeminal anesthesia due to lesions within the brain stem—to lesions affecting the end nucleus and the widely distributed roots of the fifth nerve—have a particular interest, and have already received some consideration when discussing lesions of the tubercle of Rolando. Besides anesthesia, other symptoms, which will be pointed out when lesions of the oblongata-spinal decussation are considered, occur.

The point to be remembered in this connection is that a lesion as low down as the level of the atlas gave rise to marked trigeminal anesthesia of the opposite side. In a case recorded by Stieglitz, the right cornea and right side of the face were completely anesthetic, while the right side of the body, though paralyzed, was sensible to the slightest touch, but the sense of position of the limbs was uncertain, and on the left side anesthesia to touch, pain, and temperature was complete. In this case Turner believes that the symptoms suggested a lesion above the second cervical nerve root in close proximity to the tubercle of Rolando, although it may have been higher up in the oblongata.

Anesthesia from Lesions of the Trigeminal Central Pathway. Lesions of the neurons which pass from the sensory end nucleus of the trigeminus in the oblongata to the cerebrum give rise to anesthesia or hyperesthesia according as the lesion is destructive or irritative; but I do not know of any cases of anesthesia absolutely confined to the trigeminal distribution from lesions of the internal capsule, thalamus, corona radiata, or cortex, although hemianesthesia, more or less complete, and including the face, from lesion of the posterior portion of the posterior limb of the internal capsule and of the thalamus, has been a somewhat frequent observation, and is in accord with the results of physiological experiment.

Rules for the Diagnosis of the Site of the Lesion causing Trigeminal Anesthesia. With the addition of the points just given about the occurrence of anesthesia from lesion of the tubercle of Rolando and the descending spinal root of the trigeminus, the rules long since given by Romberg for the diagnosis of the site of the lesion from the distribution of the anesthesia remain good. These are as follows: (*a*) The more the anesthesia is confined to slight filaments of the nerve, the more peripheral will the seat of the lesion be. (*b*) If the loss of sensation affect a portion of the face together with the corresponding facial cavity, one of the divisions of the nerve is affected before or immediately after its passage through the cranium. (*c*) When the entire area of the fifth is more or less anesthetic, and there are nutritive disorders in the affected parts, the Gasserian ganglion or the nerve in its immediate vicinity is the seat of the disease. (*d*) If the anesthesia of the fifth nerve is complicated with disordered function of adjoining nerves, it may be assumed that the disease is seated at the base of the brain. (*e*) If sensation is lost in the face on one side and in the half of the body and limbs on the other side, the lesion is probably situated in the lateral part of the upper extremity of the pons and preoblongata. (*f*) If sensation is lost in the face, half of the body, and the limbs of the same side, the lesion is situated in the opposite hemisphere of the brain, most probably in or near the posterior third of the posterior segment of the internal capsule.

Trigeminal Anesthesia in Chronic Degenerative and Retrogressive Diseases.—Special forms of trigeminal anesthesia are occasionally seen in connection with chronic degenerative diseases, like tabes, chronic muscular atrophy, and syringomyelia. In rare instances it may, for a time at least, be the only indication of such chronic degeneration. In a case of tabes recorded by Pierret mastication was difficult, and other symptoms present were anesthesia in the distribution of the trigeminus, impairment of speech, incoordinate movements of the tongue, tinnitus and some deafness in one ear, and constant parosmia, the patient complaining of an almost continuous bad smell. In several cases that have come under my observation trigeminal anesthesia has apparently been a portion of the syndrome of an unusual type of syringomyelia, although the possibility of syphilitic disease in these cases cannot be overlooked. In one case a man thirty-seven years old gradually lost sensation on the left side of the face. Examination showed decided loss of all forms of sensation, involving to some degree all the sensory divisions of the fifth nerve. The mucous membrane of the nose and of the mouth of the same side was also anesthetic. The patient had some sensory symptoms, chiefly paresthesias, in other parts of the body. For a short time before coming under observation he had had nocturnal headaches. His sight was poor, but this was apparently due to an old defect of refraction; no abnormal ophthalmoscopic appearances were found. The pupils were small, and the iritic and accommodation reflexes were abolished. Smell was almost entirely lost on the left side. Hearing was good. He was much troubled with drooling from the left corner of the mouth, and occasionally he had a little difficulty in swallowing. The tongue exhibited slight fibrillary tremor. Careful examination showed no motor paralysis in the domain of the fifth, seventh, fourth, or sixth nerve. In this case the trigeminal anesthesia was the dominating symptom, although the other phenomena present showed that structures outside of the trigeminal apparatus were affected. Persistent antisyphilitic treatment failed to help this patient, whose bulbar symptoms were gradually extending when he was last seen by the writer. In 1889, in association with Dr. S. Solis-Cohen, I studied another case of bulbar paralysis, with, among other symptoms, marked disturbances of sensation in the domain of the trigeminus as well as in other parts of the body.

HEMIFACIAL ATROPHY.

As stated on page 863, with the little light that has been thrown on its pathology by two autopsies, hemifacial atrophy should be regarded as a disease of the trigeminus, and should therefore be briefly considered in this connection. It is a trophic disease characterized by wasting of all the tissues of one side of the face except the

muscles. Its synonyms are unilateral atrophy of the face, progressive facial hemiatrophy, neural atrophy of the face, facial trophoneurosis, and progressive laminar aplasia. In rare instances the muscles are involved in the atrophy, but usually this is confined to the skin, the subcutaneous tissue, and the bones. The earliest description of the disease was by Parry, in 1825. Its clinical literature has grown rapidly in recent years, but thus far pathological reports have been made on only two cases, those of Homén and Mendel. The disease usually develops very slowly, commonly beginning with a whitish patch on one side of the face. Other similar patches appear, and in time change to a darker hue. The alterations in color are chiefly dependent upon the cutaneous atrophy. As a rule, the disease advances to a certain point and then remains stationary, apparently not interfering, or interfering but little, with the patient's length of life. The atrophy causes a marked lessening of the bulk of one side of the face (Fig. 412). Changes in the color of the hair take place, and even complete absence of the hair in spots has been observed. The beard may drop out or fail to grow on the affected side. In an interesting case recorded by Lloyd the patient first noticed, several years before coming under observation, a white patch near the bridge of the nose. This gradually spread upward and invaded the hairy scalp. The skin of the affected area was almost destroyed, and the epidermis left dead-white and shrivelled. Where the atrophy involved the hairy scalp the hair had fallen out, causing a most unsightly appearance. The atrophy was largely confined to the upper part of the face. This case in its location and phenomena presented some of the peculiarities of the circumscribed form of morphea, but the latter does not usually involve the bone. In this case not only had the fat and connective tissue disappeared, but the bone was also deeply atrophied. It is doubtful whether a clear distinction can always be made between morphea and hemifacial atrophy of the partial form. Sometimes the sebaceous glands are atrophied with the skin. The bones of the jaw, especially those of the upper jaw, may waste, and the teeth occasionally fall out. Other symptoms of irregular occurrence are decrease or absence of perspiration, and even of the lachrymal secretion. In rare instances

Fig. 412.

Hemifacial atrophy. (Hirt.)

disorders of taste and hearing have formed a portion of the syndrome of the disease. It is an important fact, as bearing upon the theory of the trigeminal origin of the disease, that the sufferers from hemifacial atrophy sometimes are the victims of various forms of trigeminal neuralgia, this usually being observed in limited portions of the nerve distributions. Neuralgic attacks, as a rule, come in paroxysms, and in rare cases are accompanied by twitchings of the muscles supplied by the seventh. Other symptoms referable to the trigeminus are hyperesthesia, paresthesia, and even slight anesthesia. Paralysis of the face or limbs is rare. Besides the symptoms referable to the sensory distribution of the trigeminus, in other rare cases masticatory twitchings and atrophy of some of the muscles supplied by the motor branches of the trigeminus are observed. Occasionally, instead of one half of the face being involved in the atrophic process, this is confined to a more limited area, as to the lower or the upper half of one side. Recorded cases would seem to indicate that the disease is much more frequent in the female sex, and it usually occurs before the age of twenty years, not a few of the cases originating during childhood. Conclusive facts with regard to heredity are wanting. Various local affections have been assigned as causes, as otitis, glandular inflammation, and rheumatic neuritis or perineuritis due to cold. It has been observed in association with various organic and especially with degenerative diseases of the neuraxis, as disseminated sclerosis, syringomyelia, and epilepsy. Moebius has suggested the theory of external local infection. The facts point to the fifth nerve or its nuclei as the source of this affection, although the recorded cases are not sufficient to be authoritative. The disease is probably dependent upon an irritative lesion of some portion of the fifth nerve, probably of its sensory nuclei and root fibres. In one case with autopsy, by Homén, a dural tumor compressed the Gasserian ganglion and the branches of the fifth nerve, and in another case, by Mendel, examination revealed proliferative neuritis of the left trigeminal nerve, most marked in its second branch, with central atrophy of the spinal root of the fifth and partial atrophy of the substantia ferruginea. Facial hemiatrophy in this case was associated with atrophy of the left upper extremity, and the autopsy and microscopical examination showed that the left musculospiral nerve had undergone changes similar to those found in the Gasserian ganglion and the fifth nerve. The history of an insidious onset beginning early in life, the fact that the atrophic process does not implicate, or implicates to only a slight degree, the muscular tissues, and the distinctive appearances presented by the side of the face affected, are sufficient to enable a diagnosis to be made. Asymmetry of the face and head is not uncommon in idiocy and imbecility and in other cases of arrested development, but the atrophy in these cases is not of the circumscribed character and has not the special

features of hemifacial atrophy, especially as observed in the skin. One side of the face may be smaller than the other without any real atrophy being present, and in these cases investigation will show that the various tissues composing the face are properly developed on both sides, the only difference being in general bulk and contour. Peripheral facial paralysis is readily separated from hemifacial atrophy by a study of facial movements and of the electrical reactions. In facial paralysis atrophy is usually not present, or is very slight, and the deformity shown by the face is due either to over-action of the muscles of the unaffected side or to secondary twitchings and contractures in late and not entirely cured cases. The prognosis as to recovery is bad, but the disease does not seem to shorten life. Cases have lived from thirty to forty years after the first observations upon them were made. Treatment is futile, at least so far as the records up to the present time are concerned. Thyreoid feeding may be tried, as may also the use of mild galvanic currents and massage. As in other cases of disease of a degenerative type, one of the best things that can be done for the patient is to keep up the general health by hygienic and other measures. Pads introduced into the mouth have been suggested to prevent the unpleasant appearance caused by the atrophy.

HEMIFACIAL HYPERTROPHY.

In hemifacial hypertrophy one side of the face is enlarged, owing to the increase of all its tissues, the muscles, however, being less involved than other structures. A few congenital cases have been recorded. One of the most interesting cases of acquired hemifacial hypertrophy has been recorded by Montgomery, the affection having been preceded by an abscess of the cheek. The few facts known seem to point to irritation of the fifth nerve or some of its central structures as the source of the disease, but, as in the case of unilateral atrophy of the face, this etiology cannot be regarded as established. In some instances hypertrophy of the face has developed in connection with trigeminal neuralgia. No autopsies have yet been recorded. Microscopical examination of the diseased tissues shows evidences of chronic inflammation and degeneration. The disease may begin in the gums, which may be markedly

FIG. 413.

Hemifacial hypertrophy. (Montgomery.)

hypertrophied. A marked increase of size, with other deformity, slowly takes place. In typical cases both the bone and the soft tissues are involved in the process. The skin of the face becomes changed in appearance. In several of the reported cases the alveolar processes have been enlarged. The resulting hypertrophy corresponds closely with the distribution of the fifth nerve. Sensibility is not changed, and the special senses, as a rule, are not affected. The disease must not be confounded with its opposite, hemifacial atrophy, the observer supposing that the unaffected side is atrophied and the hypertrophic side normal. The appearances of the skin and the most casual inspection will correct a case of this sort. Treatment is of even less avail than in cases of hemifacial atrophy.

FIG. 414.

Hypertrophied gum of the upper jaw in a case of hemifacial atrophy. (Montgomery.)

PARALYSIS OF THE MOTOR PORTION OF THE TRIGEMINUS (MASTICATORY PARALYSIS).

Definition and Varieties of Motor Trigeminal Disease.—Disease of the motor portion of the trigeminal nerve is either paralytic or spastic, and is usually described as *masticatory paralysis* and *masticatory spasm.* In rare instances trigeminal paralysis and spasm are associated in the same case. Paralysis of the motor portion of the trigeminus, the so-called masticatory paralysis, is a disease in which the movements of the lower jaw and some of the movements of the throat are paralyzed because of a destructive lesion involving the nerve supply to the muscles controlling these movements. The paralysis may be *unilateral* or *bilateral*, and either form may be *partial* or *complete.* It may also be an isolated, purely motor affection, or it may be associated with disease of the sensory portion of the trigeminus and of other structures.

Symptomatology.—Masticatory paralysis is shown by loss or impairment of the movements regulated by the masseter, temporal, and pterygoid muscles. When the paralysis is bilateral and complete, the lower jaw cannot be held in position (see Fig. 415). In the unilateral variety the patient experiences difficulty on one side in masticating, and in opening and closing the mouth for other purposes, but the masseter muscle of the unaffected side is sufficient to hold the jaw in position, or nearly in position, and for this reason

the paralysis may at first escape attention. In brief, the main effect of paralysis of the motor trigeminus is to interfere with the movements of the lower jaw. If the muscles degenerate, this can be determined by inspection and by manipulation of the masseters and temporals, and in these muscles loss of faradic contractility and reactions of degeneration, partial or complete, may be present. A few words should be said about the special movements regulated by each of the important muscles of mastication and the effects of their paralysis. While trigeminal motor paralysis generally affects all portions of the nerve, in rare instances nuclear disease, or disease isolated to the nerve branches supplying one muscle or one set of muscles, gives incomplete varieties of masticatory paralysis, and even when the paralysis is complete a knowledge of the particular actions of the different muscles throws light upon the peculiarities of the resulting disorder. The masseter muscle acts to draw the jaw slightly forward as well as upward, and, in consequence, when it is paralyzed the jaw not only drops but also slightly recedes. The temporal muscle acts with the masseter in closing the jaw, but its posterior fibres also draw the jaw backward, and this particular movement will therefore be impaired or lost when this muscle is paralyzed. Paralysis of the external pterygoid muscles interferes with at least three actions,—that of pulling the lower jaw forward, that of carrying the ramus of one side inward (and, of course, of the other side outward), and that of depressing the jaw. As the internal pterygoid muscle assists in closing the jaw, and in advancing it when closed, and as it also acts with the external pterygoid to draw the ramus of its own side towards the middle line, its paralysis will cause impairment or loss of these movements; but an interference with the function of the internal pterygoid muscle could probably be separately determined only in the case of the movement for advancing the jaw. As the tensor palati is probably supplied by the fifth nerve, the movements performed by this muscle should be affected in complete masticatory paralysis. In one of my cases of associated masticatory paralysis and spasm, to be presently described (see page 894), the movements of this muscle were affected, as were also those of the mylohyoid and the anterior belly of the digastric. Paralysis of the tensor tympani, also supplied by the fifth nerve, probably causes tinnitus and inability to appreciate deep tones, but clinical observations regarding paralysis of this branch of the nerve are wanting. In examining for paralysis in the distribution of the motor subdivision of the trigeminus, much aid will be given by placing the fingers of both hands over the temporal and masseter muscles of the two sides of the face, when the imperfect action or entire lack of action on the affected side can be readily determined. If the paralysis is unilateral the muscles on the affected side are flaccid during mastication, while the corresponding muscles on the other side firmly contract.

Etiology and Pathogenesis.—Nothing need be said about age, heredity, sex, and other factors usually included in a study of the etiology, as in a disease so rare as masticatory paralysis these play an unimportant rôle. The determined or the presumable causes have in the vast majority of cases been focal lesions of the trigeminal apparatus,—cortical, subcortical, capsular, pontile, oblongatal, basal, or extracranial. Peripheral neuritis is a rare cause. The most frequent lesions have been tumors or localized meningitis at the base of the brain, but disease affecting the nuclei and roots of the nerve is only a little less common. In several personal cases focal lesions of the pons and oblongata have involved the motor trigeminus, but, with one exception, other nerves and important structures have been implicated. Such nuclear and radicular disease may be of degenerative type, or it may be a focal encephalitis, a gummatous infiltration, a softening from embolism or thrombosis, a hemorrhage, an enlarged vessel, or even a sclerotic nodule. Cases, however, have been reported in which degeneration of the trigeminal nuclei has been present in association with degenerative disease attacking various parts of the neuraxis. In a case of tabes recorded by Pierret, the patient appeared to be always chewing, and he was obliged to swallow with extreme care. Speech and deglutition were difficult; the tongue on protrusion was jerky and tremulous. Later all the muscles of the eye were paralyzed. The chewing movements continued; saliva ran from the corners of the mouth; the teeth did not meet on attempting to bite or to seize objects placed in the mouth.

Diagnosis.—The diagnosis of masticatory paralysis is difficult only when it is of slight degree, and especially when it is unilateral. The points as to the special actions of the different muscles and as to methods of examination given under symptomatology should be here recalled. Frequently diagnosis will be assisted by the fact that the motor paralysis is associated with symptoms of involvement of the sensory branches of the trigeminus. Cortical or subcortical masticatory paralysis is usually associated with symptoms showing paralysis of other parts. As cortical lesions are in most cases unilateral, and as the cortical representation of the movements of each lower jaw is bilateral, that is, the centre on one side of the brain being sufficient for both sides of the body, a cortical masticatory paralysis is sometimes scarcely recognizable. Degeneration reactions are observed in cortical cases, and indeed always where the lesion is situated cephalad of the motor nucleus. In pseudobulbar paralysis from lesion of the lenticula or internal capsule, masticatory paralysis may form a portion of the syndrome. The diagnosis will be made by a study of the associated symptoms and regulated by the absence of the symptoms of true bulbar paralysis. The diagnosis of nuclear masticatory paralysis is made by the rules governing the diagnosis of nuclear paralyses of all sorts. (See nuclear facial paralyses.) Basal

meningitis, tumors, hemorrhages, and abscess nearly always involve adjacent structures, and a careful study of the focal symptoms is usually sufficient to point out the position and limitations of the lesion causing the trigeminal paralysis. Schulten, for instance, reports a case of complete paralysis of the trigeminus in its motor as well as in its sensory portion, in which case the abducens and oculomotor nerves were both involved.

Prognosis.—The prognosis of masticatory paralysis depends upon the nature, and, to a less degree, upon the situation, of the lesion when it is focal. In cases of syphilitic disease at the base, the prognosis will be relatively unfavorable, as it is also in the rare cases due to peripheral neuritis. Nuclear cases are of course unfavorable.

Treatment.—The treatment of masticatory paralysis can be reduced to a few points. It is that of focal specific disease, or local meningitis or neuritis from whatever cause. Mercury, iodides, salicylates, and hydriodic acid, the remedies so frequently referred to in these cases, are of course indicated. Electricity can be used, guided by the same rules that apply to other peripheral and central palsies. Strychnine, the hypophosphites, and other tonics are also useful.

SPASM OF THE MUSCLES SUPPLIED BY THE MOTOR DIVISION OF THE TRIGEMINUS.

Synonyms and Varieties.—Spasm of the muscles supplied by the motor division of the trigeminal nerve is known by several names, as *masticatory spasm*, *trismus*, and *masseter spasm*. The spasm may be *tonic* or *clonic*, *acute* or *chronic*, although the acute variety is rare; it may be *hysterical* or *reflex*, due in the latter case especially to irritation reflected from the sensory subdivision of the fifth. One of the varieties of clonic masseter spasm is known as *jaw chattering*. So far as varieties of the spasm due to focal lesion are concerned, reported cases show that the disease may be situated in the cerebral cortex, the corticobulbar pathway, the oblongatal nucleus, the root fibres within the pons, or in the trunk of the nerve itself.

Clinical History.—When the spasm is tonic and bilateral the jaws are held more or less firmly closed, and sometimes an actual grinding of the teeth takes place. In other cases the jaws can be partially opened by the patient, or can be forced slightly open by others, but the exercise of the force necessary to do this may cause pain, especially in the masseter muscles. When the spasm is marked, the existence of strong contraction in the bellies of the masseter and temporal muscles causes them to stand out prominently, and the muscles can be felt to be hard and bulging by passing the fingers over them. The fixation of the jaws gives the patient's face a set expression, and the expression of emotion, as of pleasure or grief, is evinced only in the action of the muscles supplied by the seventh pair and by the movements of the ocular muscles. Unilateral tonic

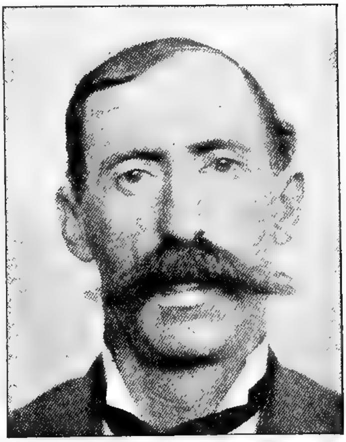
spasm is very rare, but has been recorded. Masticatory spasm even when confined to one side will hold the jaws of both sides together, but in this case less difficulty will be experienced in partially opening the mouth, especially on the side which is not spastic. Differences in the bulging and hardness of the muscles of both sides can also be made out in unilateral cases. *Trismus* in its narrowest sense is a tonic spasm of the muscles supplied by the trigeminus, and especially of the masseters, in consequence of which the jaws are held rigidly closed. In the infectious disease known as tetanus one of the chief manifestations is trismus, a symptom so important and frequent as to have given the affection its common name of lockjaw. Tetanus, however, is more properly considered either under general neuroses or microbic diseases. In it, besides the muscles of the jaw, those of the throat, of the neck, of the trunk, and even of the limbs in some cases, may be involved; it is only in rare cases that the tonic spasm is confined to the trigeminal musculature. In addition, other symptoms, like pain, great weakness, and mental excitement or depression, are present. In the first place, cases of clonic masticatory spasm are seen in which the spasmodic movements are frequently repeated, causing a disorder sometimes spoken of as chattering jaw. The chattering is almost continuous, except when the patient is asleep or during mastication. It is a symptom of senility, and is sometimes seen in paralysis agitans and in some forms of sclerosis. In one case of this kind, an old colored man in the nervous wards of the Philadelphia Hospital, the patient had, in addition to chattering jaw, some tremor of the limbs on one side, and a tendency to assume bent and fixed attitudes. Another case of clonic masseter spasm has been reported from my Philadelphia Polyclinic service by Dr. J. W. McConnell. This patient was a woman past seventy years, from whom no family or personal history could be gleaned. Six months before coming under observation she first noticed a "peculiar quivering sensation in the region of the stomach," which was followed by nausea, and sometimes by vomiting. Immediately after these symptoms a movement of the lower jaw would occur, lasting less than a minute, but frequently repeated. The woman was of worrisome disposition, and could think and talk of nothing but her troubles. Her general health was good. Her face was intermittently involved in bilateral spasm or twitching of the masseter muscles. The spasm was not attended with pain. The intervals of stoppage varied from a few seconds to almost a minute, and if her attention was diverted the period was even longer. So far as known, the movements ceased during sleep, but did not entirely stop when mastication was attempted. The spasm was irregular, and an attempt to count the oscillations was fruitless. Instead of this form of clonic masseter spasm, which occurs chiefly among the aged, a variety is seen in which spasm occurs at short and dis-

tinct intervals and may be repeated only once or twice. Still another form of clonic masseter spasm is clearly of hysterical origin, and may have some bizarre features. Verhoogen has reported the case of a boy who had received from a comrade a slap on the right cheek. The following morning when the patient awoke he could not open his mouth. When asked to open his mouth a month after the accident, he separated the lips, but the jaw remained immovable, and any attempt to separate the teeth immediately provoked severe pain in the right masseteric region. With great care and gentleness it was possible slowly to open the mouth to a reasonable extent, but during this procedure the masseters were observed to contract repeatedly. At the angle of the jaw there was an area of extreme cutaneous tenderness; on and over both arms were areas in which the thermic sense was lost. Under chloroform the spasm relaxed, and the joint was found to be absolutely normal. The contracture, then, was purely hysterical, the slight traumatism having acted simply as a determining and localizing agent. The mother was a confirmed hysteric. Peterson has recorded the history of a woman in whose case the work about the mouth, and the necessity for keeping her mouth open for a long time while in the dentist's chair, in having two sets of teeth fitted to her upper jaw, resulted in the development of masticatory spasm. When sitting quietly, not using the jaw muscles, the masseters, temporals, and pterygoids were in continuous clonic spasm, the jaw opening and shutting slightly and moving from side to side. The chief difficulty, however, was when she attempted to speak, when the mouth opened widely and a subluxation of the jaw downward and forward from the glenoid cavity took place. I do not know of any records of cases of masticatory spasm apparently due to cortical disease. As stated on page 336, Horsley has placed the centre for movements of the lower jaw just behind the laryngeal and pharyngeal centres. Irritation of this region should cause lateral and up and down movements of the lower jaw. In one case, reported by Hirt, spasm of the tongue and spasm of the lower jaw were associated. The lower jaw would first be jerked to and fro and up and down, and these movements would be followed by turning and rolling movements of the tongue, the mouth remaining open. The attack occurred ten or twenty times a day, and came on for the first time three days after an epileptic fit.

Associated Paralysis and Spasm of the Trigeminus.—In rare instances trigeminal paralysis and spasm are associated. One interesting case of this kind, studied in my Philadelphia Polyclinic service, has been put on record by McConnell. The patient was a man forty-six years old. On December 1, 1892, he was affected with symptoms of an acute cold, and about a month later he noticed for the first time that he could not chew as well as formerly. The weakness of the muscles of mastication increased, and in the course

of two or three weeks they became powerless to such an extent that the lower jaw was not held in position against the upper. Ability to bring the jaws together was variable. In the latter part of February the jaw became affected with a tremor, causing chattering or clonic spasm, and occasionally the jaws would snap together involuntarily. About the beginning of March he became unable to close the mouth except with external assistance. The notes of the case state that he was a fairly well nourished man, without any paralysis of the limbs or trunk, but with a peculiar appearance of the face. The chin is dropped, causing a separation of the teeth of about one quarter to one half inch, with the lower lip somewhat everted and pendulous. (Fig. 415.) Attempts to bring the jaws together cause tremor. Movements of the lips and other portions of the face supplied by the seventh nerve are preserved. Sensation is also preserved. The movements of the temporal, masseter, and pterygoid muscles are all greatly impaired. The patient is unable to swallow food that is masticated and at the same time hold in his mouth the part that is not masticated. In order to swallow fluids he must push his mouth shut after the drink is taken, and drop his head towards his chest. If he does not do this, what is taken will regurgitate through his nose. Once in a while fluids get into his trachea. Warm drinks are especially irritating. The act of swallowing, which was carefully studied, is very peculiar; a part of the fluid seems to go down by gravity, then a spasmodic action of the muscles of the front of the neck takes place, and some of the food or fluid is swallowed and some regurgitates through the nose. The movements performed by the mylohyoid and digastric muscles are affected, and those of the tongue seem also to be impaired. The palatal muscles contract to irritants somewhat imperfectly. The constrictors of the pharynx also contract a little less actively than usual; the vocal cords and arytenoids move perfectly. In drinking he seems to be unable to get the water far enough back for the constrictors to act. Laughing gives the upper part of his face a peculiar appearance, somewhat like the *risus sardonicus*. This case, as already stated, would seem to give some clinical support to the old view that the nerve supply to the tensor palati

Fig. 415.



Trigeminal paralysis and spasm in the same case. Besides the paralysis of the masseter, pterygoid, and temporal muscles, the movements performed by the digastric and mylohyoid muscles were impaired, as were also those of the tongue, the palatal muscles, and the constrictors of the pharynx.

is not properly from the vagus itself, but that the nerve branches come from the trigeminus, and pass by way of the vagus trunk, leaving it to go through the otic ganglion to the muscle.

Etiology.—The etiology of spasm of the trigeminal muscles is that of local spasm affecting the distribution of any of the motor cranial nerves, and, indeed, of local spasm anywhere in the body. Most cases are due to focal lesions in the motor trigeminal pathway or to diffuse lesions affecting this nerve as well as other structures. Toxic or infectious agents acting upon the centres for the nerve may be a cause in some instances. Hysterical and neurasthenic states and the feebleness of the nervous system which comes with advancing years or premature senility are other causes. Among exciting causes the most important are sudden and excessive emotion, exposure to cold, and injuries. In Peterson's case the spasm originated as the result of prolonged dental operations, and slight spasm of the muscles of the jaw after dental operations is not uncommon. Any focus of irritation in the sensory distribution of the fifth may give rise to the spasm.

Diagnosis and Prognosis.—The diagnosis is readily made. Acute affections of the maxillary articulation, or painful and especially inflammatory diseases of the jaw or of the mouth, which prevent it from being opened, might for a time be regarded as due to masseter spasm, and indeed such spasm sometimes accompanies painful disorders of this description. Brief study of the condition of the mouth and the jaws and inspection and manipulation of the masseter and temporal muscles will clear up all doubts. In one of the varieties of arthritis deformans the temporomandibular articulation, the spinal joints, and, indeed, the articulations of any part of the body, may be rigidly fixed, but the mistake of supposing such a case to be one of tonic masticatory spasm could be made only by the most careless observation. The prognosis varies according to the nature of the affection: it is best in hysterical cases, in those due to irritation reflected from sensory nerves, and in cases of organic lesion that can be favorably influenced by absorbents. When the nuclei or root fibres of the nerve are involved in gliomatous or degenerative processes the prognosis is, of course, bad; such cases progress rapidly or slowly to a fatal issue.

Treatment.—The treatment of trigeminal spasm must, of course, be based upon the nature of the cases. If due to a focal lesion at the base of the brain or elsewhere in the cranial cavity, or to a diffuse inflammation, absorbent remedies, like the iodides, mercury, and ammonium chloride, will be indicated. The possibility of a reflex origin of the spasm should always be borne in mind, and sources of irritation should be sought for, especially in the distribution of the sensory portion of the fifth nerve. The nasal, aural, and pharyngeal cavities should be searched, the teeth and gums should be examined

for caries or other disease, and the presence of periosteal inflammation or of trouble at the inferior maxillary articulation should be determined. In rheumatic cases the salicylates and other antirheumatic remedies should be used. For the relief of the spasm, especially in tonic cases, various antispasmodics and sedatives may be used. Those which have proved of most value are atropine, hyoscine, coniine, and duboisine. In Peterson's case, duboisine sulphate in doses of one two hundredth of a grain three times daily gave the patient much relief by quieting the spasmodic movements almost entirely. Coniine hydrobromate and fluid extract of gelsemium are remedies worthy of trial, and should be pushed rapidly to maximum doses until constitutional effects are produced. Beginning, for example, with one twenty-fifth of a grain of coniine hydrobromate four or five times daily, the dose can be increased until one tenth of a grain is taken, and the fluid extract of gelsemium can be pushed until from ten to fifteen minims are taken at one time. In the administration of these drugs, for this as for other diseases, it is of course important to watch carefully the effects produced and to study the idiosyncrasies of the patient. Bromides have a decided temporary effect on the spasm, especially when it appears in a periodical or paroxysmal form of clonic spasm. The use of the actual cautery is of great value here, as in many other forms of local spasm. It should be applied either to the back of the neck or to one or both sides of the face over the insertion of the masseters. The best method of using the cautery is quick, small burns somewhat frequently repeated, as two or three times a week. The application can be made at a certain spot on one day, and subsequently on both sides of it and near it until the first burn is entirely healed. Peterson had made especially for his case an apparatus which kept the patient's jaw closed and allowed her to talk between her teeth without the uncomfortable tonic spasm of the depressors of the jaw, although the clonic movements of the masseters and pterygoids continued. Such an appliance may be of service in other cases. The question of the best method of feeding the patient in a case of serious tonic spasm may become one of importance. This can be accomplished by the old method of extracting a tooth if one or more of the teeth have not already been removed, or the feeding may be accomplished with the nasal tubes similar to those used for the insane, although in nasal feeding the passage of the tube might in some instances give rise to palatal or pharyngeal spasm. Nerve stretching or resection, and myotomy, have been suggested, but no facts have been furnished which show their value. The only justifiable operation with our present knowledge would be stretching of the motor subdivision of the fifth, if this could be separately reached. The nerve can be reached within the cranium by the same procedures that are employed in exposing the Gasserian ganglion.

CEPHALIC TETANUS.

In *cephalic tetanus* the phenomena are so definitely related to trigeminal irritation that, although the disease may be primarily due to a general infection, it seems best that it should be briefly considered. It is designated by other names, as *head tetanus* (*kopftetanus*) and *tetanus hydrophobicus.* It is usually the result of a wound somewhere in the distribution of the fifth nerve, and the chief symptoms are trismus and paralysis of the face on the same side. In some cases the muscles of the nonparalyzed side of the face, the muscles of the throat, and the respiratory muscles are also affected with spasm, and in other rare instances the spasm, at first confined to the trigeminal distribution, spreads to all parts of the body, the case thus becoming one of the ordinary type of tetanus. Up to 1895 only three American cases of cephalic tetanus had been placed on record. Accounts of these and the entire literature of cephalic tetanus have been summarized by Willard and Johnston, who give a table of no less than seventy-five contributions on this subject. The notable features in the case of Willard and Johnston were that the patient suffered from general traumatic tetanus, recovering after an illness of about two months; that associated with it was a hemifacial paralysis which disappeared with recovery from the tetanus; that the wound was an insignificant one, but involved the filaments of one of the branches of the fifth nerve; and that decided improvement in all the symptoms followed subcutaneous section of the nerves in the injured area. While cephalic tetanus may follow an injury to any of the cranial nerves, it is more frequently found after such injury to the orbital and nasal branches of the trigeminus. Like other forms of tetanus, it is in all probability microbic in origin. The influence of the tetanus bacillus in the production of the disease seems to have been thoroughly demonstrated by a number of observers who have isolated the pin-shaped bacillus. The cause of the facial paralysis in cephalic tetanus has given rise to considerable discussion. In the case of Willard and Johnston, careful investigation was made to exclude the possibility of its being merely a coincidence due to independent causes, and the absence of middle ear disease, intracranial disease, brain abscess, etc., was thoroughly demonstrated. These writers believe that in the present state of our bacterial knowledge it is most rational to refer this paralysis to a direct toxic effect of the poison acting upon the filaments of the seventh and fifth nerves. The mortality in acute cases is as high as ninety per cent. In chronic cases—that is, cases in which the symptoms arise in the first week after the wound and the affection develops somewhat slowly—the prognosis is much better. Of thirty-two chronic cases collected by Willard and Johnston, twenty-four recovered and eight died. The treatment is much the same as that for tetanus of any variety.

DISEASES OF THE SEVENTH OR FACIAL NERVE.

General Description of the Facial Nerve and its Central Apparatus.—The seventh (*facial nerve, portio dura* of the *seventh nerve, nervus communicans faciei*) is a purely motor nerve. It is described differently by different anatomists.* It is, for instance, sometimes described as composed of a main part, the facial nerve proper, and an accessory portion, the *pars intermedia of Wrisberg*. The views held by the writer with regard to the latter have already been given at length on pages 691–693, where it has been shown that the facial nerve and the intermediary nerve of Wrisberg with its peripheral extension (the chorda tympani nerve) have a segmental companionship similar to that which exists between the motor and sensory spinal nerves; that the true origin of the pars intermedia and chorda tympani is in the geniculate ganglion, and that the former has a separate end nucleus in the preoblongata. The paralytic and spasmodic affections involving the distribution of the facial nerve, like the disorders of other cranial nerves, are best understood by keeping in mind the entire facial encephalic apparatus. Under cortical localization the position and subareas of the cortical facial centre are shown and described. The various movements of the muscles of the face supplied by the seventh nerve are represented around the foot of the central fissure. The central neurons of the facial apparatus, therefore, arise from the motor cells of the lower extremities of the central convolutions. Together they form a tract which passes by the corona radiata and internal capsule about its knee to the tegmentum, decussating in the latter to enter the facial nucleus of the other side. From this nucleus by a tortuous intraencephalic and extraencephalic course the nerve passes peripherally. (Fig. 416.) A probable connection of the facial nerve with the thalamus should not be overlooked, although, as this has not been fully traced, it is not indicated in the scheme. Some observations indicate that if the connection of the cortical facial centre with the seventh nucleus is broken, voluntary innervation of the muscles supplied by the seventh nerve on the same side does not take place, but emotional innervation through pain or laughing, as this is more of a reflex nature, is possible. If one thalamus is destroyed while the control tract of the seventh through the internal capsule from the cortex and pons remains intact, the voluntary innervation of the facial muscles can be accomplished, but the involuntary, as in laughing and crying, is interfered with on the same side. It would seem from these observations that the facial nucleus may be closely connected with the thalamus, and that by this tract the psychic reflex may be completed.

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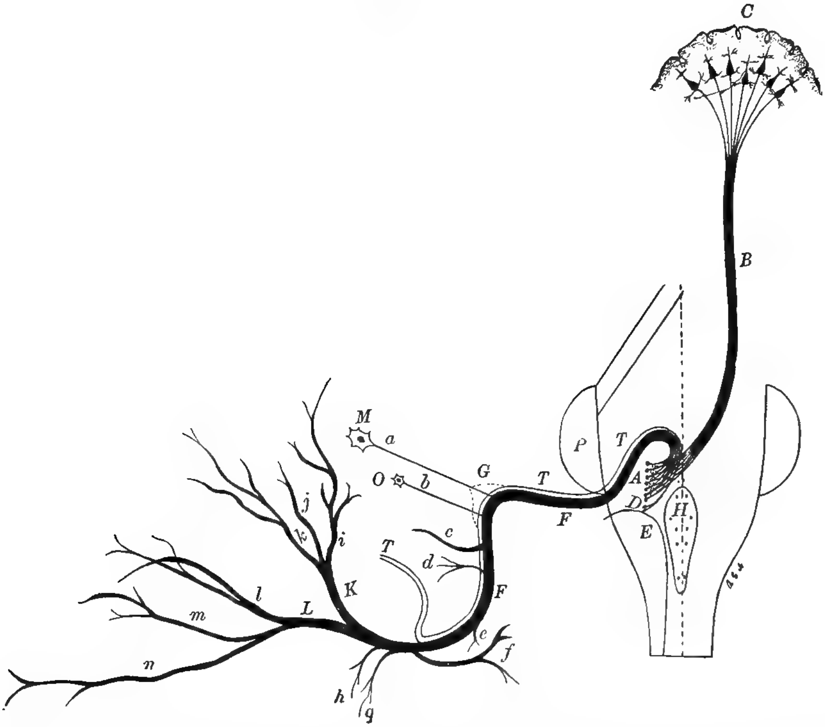
* The seventh pair of Willis includes the *portio dura* and the *portio mollis*,—the former being so called because of its hardness as compared with its companion the auditory nerve.

Nucleus and Root Fibres.—The nucleus of the facial nerve, about four millimetres long, is situated in the ventral portion of the tegmentum, four or five millimetres beneath the floor of the fourth ventricle to the mesal side of the descending spinal root of the trigeminus and the anterior root of the auditory nerve, immediately

FIG. 416.



Scheme of the apparatus of the facial nerve: *P*, pons; *A*, facial nucleus; *B*, facial corticobulbar tract; *C*, cortical centre for facial movements; *D*, nucleus of the pars intermedia of Wrisberg; *E*, descending glossopharyngeal roots; *H*, nucleus of the hypoglossal nerve; *FF*, trunk of the facial nerve; *K*, temporofacial branch of the facial nerve; *L*, cervicofacial branch of the facial nerve; *G*, geniculate ganglion; *TTT*, pars intermedia of Wrisberg and chorda tympani nerve; *M*, Meckel's sphenopalatine ganglion; *O*, otic ganglion; *a*, great superficial petrosal and Vidian nerves; *b*, lesser superficial petrosal nerve; *c*, external superficial petrosal nerve; *d*, branch of the facial nerve to the stapedius muscle; *e*, branch of the facial to the auricular branch of the vagus; *f*, posterior auricular branch; *g*, digastric branch; *h*, stylohyoid branch; *i*, temporal branch; *j*, malar branch; *k*, intraorbital branch; *l*, buccal branch; *m*, supramaxillary branch; *n*, inframaxillary branch.

dorsad and laterad of the superior olive. It is composed of multipolar nerve elements. Rootlets leave this collection of cells and come together at the floor of the ventricle in a compact bundle which has a remarkable course. Curving over the abducens nidus from behind, it overlies it like a horseshoe hung over a ball; not as a straight but as a bent horseshoe, bent so that its cephalic (efferent) branch is pointed more laterad than its caudal branch, which is the one that

emerged from the nidus. (Spitzka.) The curve is at the same time convex mesad. This part is known as the *genu facialis*, or knee of the facial, and must not be confounded with the knee of the peripheral part of the facial within the Fallopian aqueduct. After having passed around the abducens nidus from below upward, the facial roots turn obliquely outward, and, passing through the tegmentum and transverse fibres of the pons, reach the surface in a position relatively between the acoustic and abducens nerves. Recent observations of Ramón y Cajal are in accord with those of others in showing that fibres belonging to the seventh nerve decussate in the raphe behind the dorsal longitudinal bundles. He believes that the facial nucleus receives axis cylinders from the cells of the substantia gelatinosa, which substance accompanies the descending spinal root of the fifth, connection thus being made between the fifth and seventh nerves. The collaterals of these axis cylinder processes from the cells of the substantia gelatinosa are numerous. Mendel found in newborn rabbits degeneration in the posterior part of the third nucleus after destruction of muscles supplied by the upper branch of the seventh, and believed that this branch arises in the posterior part of the third nucleus and is connected through the dorsal longitudinal fasciculus with the knee of the seventh nerve. Turner, speaking of this supposed connection between the hind part of the third nucleus and the issuing root of the facial nerve, says that he has observed in two cases, in which the facial nucleus was degenerated and the oculomotor nuclei were healthy, a large number of normal nerve fibres passing into the otherwise atrophied facial nerve root from the opposite dorsal longitudinal bundle, an observation which has been confirmed by a few other observers in similar cases. A similar connection is stated to exist between the hypoglossal nucleus and the facial nerve, but direct anatomical evidence of this is thus far wanting, although some clinical facts lend support to the view. Minor connections are said to exist between the facial nucleus and the corpus trapezoides and the sensory trigeminal root. (Turner.)

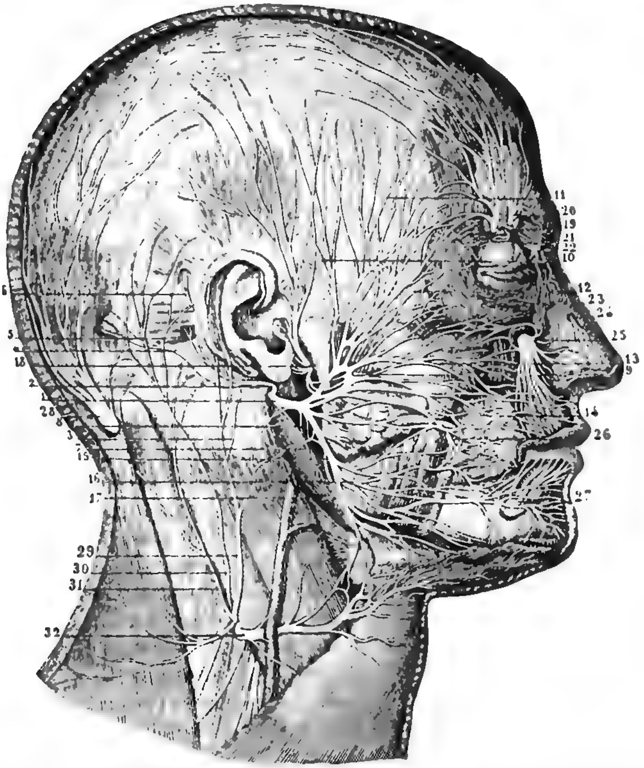
Peripheral Course of the Facial Nerve.—*Subdivisions and General Course.* The facial nerve may be conveniently subdivided into (1) an intracranial portion, (2) an intraosseous portion, and (3) an extracranial portion. The intracranial portion reaches from the superficial origin of the nerve in the groove between the olive and the restis, at the junction of the pons and the postoblongata, to its point of entrance into the internal auditory meatus; its intraosseous course begins in the internal auditory meatus, but is almost entirely confined to the winding route of the Fallopian aqueduct; while its extracranial course is usually given as first from the stylomastoid foramen through the body of the parotid gland, after which it subdivides into an upper temporofacial and a lower cervicofacial branch. Some anatomists state that it runs between the parotid gland and the ante-

rior border of the sternocleidomastoid muscle. Dominguez y Raldán, after making a careful study of facial paralysis, concluded that the facial nerve did not penetrate the parotid gland. This observer made several other interesting observations regarding the anatomy of the extracranial portion of the nerve. He found that it extends a distance of two centimetres in the space called the stylomastoid infundibulum; that the superior part of the infundibulum is formed by the widening of the Fallopian conduit; and that in this part of the infundibulum the facial nerve is surrounded by a dense cellular tissue and by a lymphatic space.

Relations, Connecting Branches, and Distribution. Within the cranium the relations of the facial nerve to other parts are readily traced and described. The abducens nerve at its origin lies to its inner or mesal side, the eighth nerve to its outer or lateral side, while between the facial and the auditory nerve is the slender pars intermedia of Wrisberg. It passes forward from the junction of the medipeduncle (*crus cerebelli*) with the postoblongata, and is in relation with the auditory nerve until it reaches the internal auditory meatus, where these two nerves part company. Within the Fallopian aqueduct important nerve branches are given off, while others communicate with or lie in contact with the facial. Soon after its entrance it passes outward and forward through the bone above and between the cochlea and the vestibule, making then a sharp, almost rectangular bend, which constitutes the so-called knee of the facial (*genu nervi facialis*). From this point it proceeds backward and slightly downward to its exit at the stylomastoid foramen. At the position of the genu is a gangliform swelling (*ganglion geniculi, intumescentia gangliformis, geniculate ganglion*), from which spring the pars intermedia and chorda tympani. The three superficial petrosal nerves all communicate with this ganglion. Distally to the geniculate ganglion a branch to the stapedius muscle is given off. From the geniculate ganglion the chorda tympani passes with the facial until it branches off to join the lingual nerve, and secretory nerves pass with the chorda tympani to go to the salivary glands. A communicating twig to the pneumogastric is also given off in the descending course of the facial before it reaches the stylomastoid foramen. Just after the nerve emerges from the Fallopian aqueduct at the stylomastoid foramen it gives off a posterior auricular nerve, and a little later it communicates with the auricular branch of the pneumogastric. It supplies the retrahens aurem, sends a slender twig upward to the attollens aurem, and ends in a long, slender branch which passes backward to supply the posterior belly of the occipitofrontalis. The facial nerve supplies all the muscles of the face except the ocular muscles and the muscles which receive their supply from the motor portion of the fifth nerve. It is hardly correct to say, as is often stated, that it is the motor nerve of all the muscles of expression in the face, as the masseter and even

other muscles supplied by the trigeminus, and the ocular muscles also, are certainly concerned with facial expression. It supplies the occipitofrontalis and platysma muscles; the muscles of the eyelids, except the levator palpebræ and the unstriated muscular fibres; the muscles of the eyebrows; the extrinsic muscles of the ear; the mus-

FIG. 417.



The facial nerve: 1, trunk of the nerve after emerging at the stylomastoid foramen; 2, its posterior auricular branch; 3, anastomosis of the latter with the great auricular nerve of the cervical plexus; 4, 5, 6, branches to the contiguous muscles; 7, 8, branches of the facial to the digastric and stylohyoid muscles; 9, temporofacial division of the nerve; 10, branch to the temple, anastomosing with the auriculotemporal nerve; 11, temporal branches; 12, 13, infraorbital branches; 14, 15, cervicofacial division of the facial nerve; 14, buccal branches; 16, mandibular branches; 17, cervical branches; 18, auriculotemporal nerve; 19, 20, supraorbital nerves; 21, terminal branch of the lachrymal nerve; 22, infratrochlear nerve; 23, branch of the temporomalar nerve; 24, external branch of the nasal nerve; 25, infraorbital nerves; 26, anastomosis between the buccal branch of the inferior maxillary nerve and the buccal branches of the facial nerve; 27, mental branches of the inferior dental nerve; 28, great occipital nerve; 29, 31, branches of the great auricular nerve; 30, small occipital nerve; 32, superficial cervical nerve, anastomosing with the facial nerve. (Hirschfeld-Sappey.)

cles of the nose; and the muscles of the mouth,—orbicular, transverse, angular, radiating, and labial. It also sends branches to the posterior belly of the digastric and stylohyoid muscles. According to some authorities, it supplies the tensor tympani through the otic ganglion and the levator palati and azygos uvulæ; but it is more

correctly held that the supply of the tensor tympani is from the fifth, and that of the levator palati and azygos uvulæ from the vagus by branches which pass for a short distance with the facial. The branches of the facial nerve are shown in the two illustrations Figs. 416 and 417.

PARALYSIS IN THE DISTRIBUTION OF THE FACIAL NERVE.

Definition, Synonyms, and Varieties.—Paralysis in the distribution of the seventh or facial nerve is an affection, usually peripheral, which involves all the muscles of the face except those supplied by the trigeminus and ocular nerves, and also the other muscles not of the face which have been described as supplied by the facial nerve. Among the synonyms of facial paralysis are Bell's palsy, prosopoplegia, hemiplegia facialis, and mimetic facial paralysis. It is one of the most frequent and therefore one of the most important forms of paralysis. It may be *unilateral* or *bilateral*, although the latter form is very rare, and it may be *complete* or *incomplete*. We may have a number of special varieties, according to the exact site of the lesion in the facial apparatus, and it will be most convenient to consider these first.

Clinical History of Different Types of Facial Paralysis.—*Cerebral Facial Paralysis, Cortical and Subcortical.* While peripheral facial paralysis of a nearly complete type, due to lesion within the Fallopian aqueduct, is by far the most frequent form of this disease, a facial paralysis may be due to a lesion situated anywhere in the facial apparatus, as delineated in the scheme Fig. 416. It may, in other words, be cortical, subcortical, capsular, pontile, nuclear, or radicular; and after the nerve has emerged from the brain stem it may be one of several distinct peripheral varieties, according as the situation of the lesion is in the intracranial, intraosseous, or extracranial portion of the nerve.* When facial paralysis is of cortical origin it is usually associated with paralysis of the limbs and with orolingual paresis or aphasia; but rare cases of cortical facial monoplegia, and even of cortical paralysis, limited to particular portions of the facial nerve supply, have been recorded. In both cortical and subcortical facial paralysis the lower portion of the face is chiefly involved, but it is not true, as is sometimes stated, that the muscles of the upper distribution of the facial entirely escape; indeed, the oculofacial group of muscles is nearly always to some degree weakened. It is said to be characteristic of the cerebral types of facial paralysis that, although the patient is unable to keep his eyes closed, he winks reflexly, and although the voluntary movements of the mouth are paralyzed on one side, the emotional movements are usually retained,

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* Turner has presented in an excellent manner in the Edinburgh Hospital Reports, vol. iv., 1896, the diagnostic features of facial paralysis from focal lesions.

while in the peripheral type both are abolished. Little difference exists between cortical and subcortical cerebral facial paralysis. The subcortical type is extremely rare, and yet cases have been reported due to foci of disease either in the centrum semiovale or in the internal capsule. Parisot, as stated on page 572, has described a case of facial monoplegia, the only muscle involved being the depressor of the left angle of the mouth, in which the lesion was confined to a limited portion of the internal capsule just posterior to its knee. In both the cortical and subcortical types the muscles do not waste, or waste but slightly from disuse, and the electrical reactions are not changed. In facial pseudobulbar paralysis (see p. 353) the weakness of the upper facial group of muscles is not complete, so that the characteristic lagophthalmus of peripheral facial palsy is not seen.

Facial Paralysis due to Pontile Lesions. A lesion in the upper portion of one side of the pons may cause facial paralysis which will have some of the features of a cerebral monoplegia, but usually also others which enable a focal diagnosis to be made. As the descending facial tract has not yet decussated, the paralysis of the face will be on the side opposite to the lesion, and it will be associated with other phenomena according to the extensions and, to some degree, the character of the lesion. Usually some contralateral paralysis of the limbs is present, because of implication of the pyramidal tract. Conjugate deviation of the eyes or irregular impairment of ocular movements and some sensory involvement are often also present. In the section on small focal lesions of the pons and preoblongata the parts involved in such a lesion and the resulting symptoms are indicated. When the lesion is in the lower part of the pons, it is usually so situated as to involve both the nucleus and the root fibres of the facial, and gives one of the forms of alternate hemiplegia, the facial nerve being paralyzed on one side and the limbs on the other. Not uncommonly also the abducens nucleus and radicles are included in the lesion, and hence paralysis of the sixth nerve and conjugate deviation of the eyes to the side of the palsy are frequent symptoms.

Nuclear Facial Paralysis. Nuclear facial paralysis almost invariably is bilateral or becomes so. Most frequently it is a portion of the syndrome of some of the forms of bulbar paralysis, and the bulbar involvement may or may not be associated with evidences of degeneration in the spinal cord. The facial nidi, like the nidi of the other cranial nerves, may be attacked in cases of progressive muscular atrophy, amyotrophic lateral sclerosis, tabes, and syringomyelia. While completeness of paralysis, and the involvement of the upper as well as the lower fibres of the facial, are not entirely opposed to nuclear disease, complete nuclear paralysis comes on late, after long continued degenerative processes, and not suddenly or rapidly, as in peripheral cases. It develops insidiously, the muscles gradually weakening and wasting, and commonly without a preceding history

of rheumatism, injury, ear disease, tuberculosis, or other cause of gross lesion. Both sides are likely to be attacked uniformly. In nuclear cases the reflexes may be increased until the disease has nearly destroyed the facial nucleus, or the curious phenomenon known as crossed reflex action may be present. Turner has seen weakness of the orbicularis palpebrarum in three cases of nuclear ophthalmoplegia, in one of which the frontalis was also so much paralyzed that the patient was unable to frown, and he refers to several similar cases recorded by other observers. Congenital cases of bilateral paralysis of several cranial nerves, including the facial, have been reported and attributed to the lack of nuclear development.

Peripheral Facial Paralysis due to Intracranial Basal Lesions. The symptomatology of complete or nearly complete peripheral facial paralysis—the most usual and therefore the most important type of this affection—will be presently given in detail, but before doing this a few words need to be said about the manner in which the symptoms are modified according as the lesion is situated in the intracranial, intraosseous, or extracranial portion of the nerve, or in a more or less limited part of each of these peripheral subdivisions. A lesion at the base of the brain, usually a tumor or a gummatous meningitis, may involve the intracranial course of the facial. Such gross intracranial lesion will be likely to affect, in addition to the facial nerve, the eighth pair, the nerve of Wrisberg, and the medipeduncle, the pyramidal tract and the abducens being more rarely involved. The symptoms are those of complete peripheral palsy, with the addition of others like deafness, loss of taste, vertigo, and forced movements, and possibly hemiplegia of the opposite side and abducens paralysis of the same side. Some good authorities hold that loss of taste is not present in these cases, but I believe that it is probably present and sometimes escapes observation. If the pars intermedia is a portion of the gustatory apparatus—the central continuation of the chorda tympani—it will almost certainly be implicated in such a lesion. Occasionally double facial paralysis from intracranial lesion, and especially from a gummatous meningitis of the posterior fossa, is observed. In these cases the most striking association is that of bilateral deafness with the diplegia facialis. With regard to the involvement of the vestibular nerve (or crus cerebelli) I have had a few experiences showing the occurrence of marked vertigo in cases of intracranial peripheral facial paralysis. In one case, in which the symptoms pointed to a syphilitic tumor at the base, the patient had suffered from severe head pains with slight intermission for two years, when total paralysis of the left side of the face made its appearance, accompanied by loss of hearing and noises in the ear of the same side. Spells of giddiness of a peculiar character came on with the paralysis. On attempting to walk she would soon be compelled to run, and on getting into a run she would fall

on her face, unless she could stop herself by catching hold of some object. She felt as though she and the floor under her were going around. These vertiginous spells gradually grew less frequent and severe as the patient's general health improved. For three months she had double vision. For six months she had some difficulty in swallowing, fluids being sometimes regurgitated through the nose. The uvula pointed slightly towards the right, and the velum hung lower on the left than on the right side. On touching it with a probe it was retracted upward and towards the right. Taste was generally defective, but it could not be made out distinctly to be abolished on the anterior part of the left half of the tongue. She complained of dryness of the mouth. Smell was defective, but no differences could be made out between the paralyzed and the healthy side. Sensation, as determined by the esthesiometer and the faradic battery, was slightly but undoubtedly diminished, and on the affected side both faradocontractility and galvanocontractility were much diminished. Reflex movements could not be produced by irritating the skin of the face.

Facial Paralysis due to Lesions in the Intraosseous Portion of the Nerve. In considering lesions of the intraosseous portion of the facial it will be necessary to take into consideration the functions of the pars intermedia of Wrisberg, the geniculate ganglion, the various petrosal nerves, the stapedius nerve, and the chorda tympani. For most practical purposes the symptoms of intraosseous facial paralysis can be considered with reference to two subdivisions of the nerve, namely, one from the intracranial opening into the Fallopian aqueduct to and including the geniculate ganglion, and the other from the geniculate ganglion to the stylomastoid foramen. When the nerve is involved between its entrance to the aqueduct and the geniculate ganglion, the chief symptoms will be those of complete facial paralysis, including loss of taste. According to Erb, paralysis of all external branches of the facial, abnormal acuteness of hearing, and disturbance of the sense of taste, with paresis of the velum palati and abnormal dryness of the mouth, indicate that the lesion is in the vicinity of the ganglion geniculi. Althaus has divided the Fallopian aqueduct into three parts: (1) an external section, which is below the origin of the chorda tympani; (2) a middle section, which includes the chorda tympani and the stapedius nerve; and (3) an internal section, which embraces the geniculate ganglion, where the large petrosal nerve is given off, and also near at hand the small and external petrosals. When the nerve is involved between the geniculate ganglion and the giving off of the chorda tympani the symptoms will be complete peripheral paralysis, with loss of taste and special auditory symptoms if the nerve to the stapedius muscle is also involved. The involvement of this nerve causes some abnormal acuteness of hearing. As shown on page 708, contraction of the stapedius muscle

decreases the tension of the membrane of the tympanum and labyrinthine pressure. Its paralysis, therefore, would prevent relaxation and cause hyperacusis because of the abnormal membranic tension.

The Sense of Taste in Facial Paralysis. The subject of taste has been so fully considered in the section on affections of taste that it will be only necessary to refer here very briefly to the connection of facial paralysis with loss of taste. The views of the writer, as already stated, are that the nerves of taste are derived from the nerve of Wrisberg and the glossopharyngeal nerve, and that the fifth nerve is in no sense a true nerve of taste, as is held by many. It is quite certain, however, as has already been pointed out, that the facial nerve, in its course within the Fallopian aqueduct, contains fibres which have to do with taste, but we do not believe with Turner and Ferguson that it is proved that the taste fibres for the anterior two thirds of the tongue pass through the Vidian and great superficial petrosal nerves to the facial trunk, from which they pass by way of the chorda tympani to the lingual branch of the third division of the trigeminus. The fact that the chorda tympani lies alongside of the facial in part of its course is all that is necessary to be known to explain the occurrence of disorders of taste in lesions of the facial trunk. When loss of taste is present in nuclear facial paralysis it is highly probable that the terminal nucleus for the nerve of Wrisberg, or the nucleus of the gustatory division of the glossopharyngeus, is involved in the lesion.

Facial Paralysis due to Lesions external to or just within the Stylomastoid Foramen. The chorda tympani is given off about one quarter of an inch within the stylomastoid foramen, and it is possible to have a paralysis of the facial from a lesion not extending farther centrad than this point. In such a case the symptoms would indicate paralysis of all the external branches of the facial and of the posterior auricular nerve, without loss of taste. When the nerve is attacked at or just outside of the foramen the paralysis will simply involve all the external branches of the facial.

FIG. 418.

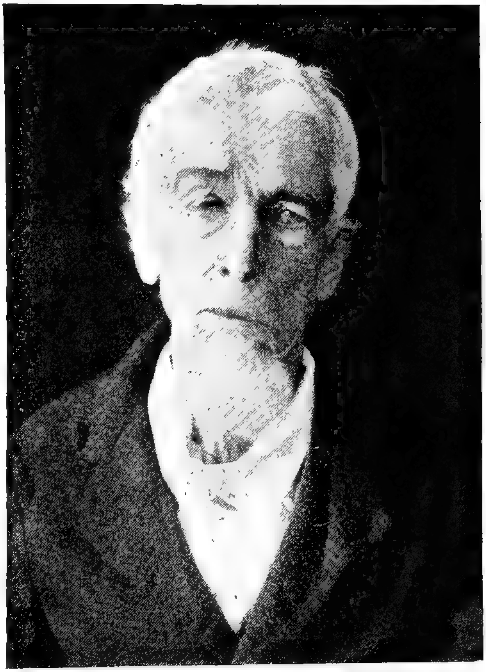
Paralysis in the upper distribution of the seventh nerve.

Paralysis limited to Special External Branches of the Facial Nerve. A paralysis limited to the temporofacial or temporocervical distribution of the facial or even to some one of its branches is occasionally observed, although such an affection is extremely rare. These cases are usually traumatic. In one of my cases the patient was a young Italian who was stabbed in front of the right ear, causing paralysis of the occipitofrontalis and paresis of the orbicularis palpebrarum of the same side. Another patient, who was struck on

the head in a brawl, had paralysis of the occipitofrontalis and orbicularis palpebrarum, and with this were associated symptoms of severe cerebrospinal traumatism. A third patient, a little boy, had fallen from a height, striking on his right temple and the side of his head. Examination showed complete loss of power in the right half of the occipitofrontalis muscle, but no paralysis of the palpebrarum or of any other muscles of the eye and face. In consequence of this loss of power there was inability to raise the eyebrow of the right side, with an absence of the natural arch of the brow, which was a perceptible distance below the line of the left. The expression of the face was that of a one-sided frowning. (Fig. 418.)

Symptoms of Complete Peripheral Facial Paralysis (Bell's Palsy). In a typical case of Bell's palsy all the muscles of the face supplied by the seventh nerve are completely paralyzed. One side of the face has in consequence a drooped, smoothed out, immobile appearance (Fig. 419). The contours of the cheek and the angle of the mouth, prominent in health, nearly disappear. On attempting to talk, laugh, or frown, the contrast of the two sides of the face is striking, giving a grotesque and unnatural look. The absence of furrowing in the forehead is conspicuous, as is also drooping of the eyebrow and the lower lid. These evidences of paralysis, which are chiefly afforded upon simple inspection, vary considerably in different cases.

FIG. 419.



Typical facial paralysis, or Bell's palsy.

Great differences in the transverse furrowing of the forehead when the face is at rest and when it is in motion are observed. In the young, in many females, and often in men who use the frontalis muscle but little, the difference in furrowing so marked on the two sides in most cases of facial paralysis is scarcely noticeable, although it will always come out to a greater or less extent on testing the patient by having him look upward. With regard to the state of the muscles no mistake need be made in a case of Bell's

palsy if a little care is taken in the examination of the patient. This examination should be made systematically, bearing in mind the numerous muscles both on the outside of the face and elsewhere which are supplied by the seventh nerve. The patient should be put through a series of volitional exercises, such as looking upward without moving the head; closing the eyes separately and together; drawing the mouth upward and outward, or in both directions at the same time; dilating the nostrils separately, which, however, is not always possible even in normal individuals; puckering the lips; puffing or blowing; and depressing and pulling outward the lower lip. We must go a step farther, and examine as to taste, as to the state of the palatal muscles and of the uvula, and as to hearing. The electrical reactions in facial paralysis are of importance, and will be further considered under diagnosis. At the very beginning, even of a severe case, the electrical reactions may be retained, but in a few days faradocontractility decreases rapidly and usually in about a week has disappeared. Changes to the galvanic current can also be noted early. At first galvanic excitability is somewhat increased; soon changes both modal and serial (page 194) are present. The order of the response to the cathode and anode may be reversed, or various changes in the reaction formula may be demonstrable.

Bilateral Facial Paralysis. Bilateral facial paralysis, or double facial paralysis, is comparatively rare. In the complete type all the muscles supplied by the portio dura are involved on both sides. The affection may be peripheral, nuclear, cortical, or subcortical. Probably the nuclear variety is of the most frequent occurrence, but when observed it is usually in association with other forms of paralysis or with evidences of degenerative disease in other parts of the body. Some years since, a case of double facial paralysis attended the service for nervous diseases of the Philadelphia Polyclinic for several months. The right side of the face was first attacked, the trouble beginning with pain and sensitiveness in the face. In about two weeks the left side of the face was also paralyzed, and the patient became deaf in the ear of the left side. The symptoms in cases of double paralysis are simply the duplication of those phenomena which have been described as present on one side; but, owing to the fact that the muscles of both sides are involved, certain peculiar conditions are brought about, as was illustrated in the case of this patient. In the first place, all the muscles of the external surface of the face supplied by the seventh being paralyzed on both sides, the face assumes a curious mask-like appearance. Whatever emotions the patient may experience, the expression will have the same dull, monotonous, unchanging look. Even the appearance of the eyes and ocular movements fail to relieve this expression, demonstrating that facial ex pression is far less dependent upon the eyes than is supposed. The patients, at least in some stages of the disorder, have great difficulty in

chewing and swallowing food. This patient, for instance, was obliged to place her hands against and around her lower jaw and cheeks in eating to prevent the food from falling out; she had also some difficulty in swallowing, and was obliged to throw her head far back to assist in this act. (Bassette.) The electrical reactions in double facial paralysis vary as in the unilateral type, according as the affection is cortical, corticobulbar, nuclear, or peripheral. In typical peripheral cases the nerves and muscles fail to respond to the faradic current, and if the case is of a serious character will give complete degeneration reactions; in the nuclear cases the reactions are those of partial degeneration. In complete bilateral palsy, according to Turner, there is no palatal movement on using the vowel sound "ah," and the pronunciation of words requiring closure of the nasopharynx is rendered imperfect; hence "rub" is pronounced "rum," and "egg" "eng." Turner here probably refers to those cases of double facial paralysis in which the lesions are situated between the geniculate ganglion and the superficial origin of the nerve.

Special Features in the Symptomatology of Facial Paralysis. Reflex movements are abolished in facial peripheral paralysis. Reflex contraction may occur in the paralyzed muscles, either through the fifth nerve by touching the skin or eyelashes, or through the optic nerves by making a rapid movement towards the eyes. While secretory disorders of the face are not manifest, it has been found that subcutaneous injection of pilocarpine does not cause sweating of the paralyzed side. Spontaneous movements, not of the nature of secondary spasms, may occur in the muscles during recovery. Sometimes associated movements occur in the paralyzed muscles. When, for instance, an attempt is made to close the eye, the angle of the mouth is drawn outward and upward, and, conversely, when an attempt is made to draw the angle of the mouth to one side, the eyelids contract. (Ross.) What are called the secondary contractures of facial paralysis are among the most annoying and intractable symptoms of old cases of this disease. These, although known to the older observers, were first minutely described and discussed by Hitzig. Commonly they appear first in the third or fourth month in the form of a slight tonic contraction in the muscles of the mouth. Eventually the muscles of the paralyzed side become the seat of almost persistent tonic spasm, more or less frequent attacks of twitching or clonic spasm occurring at irregular intervals. In time the face becomes spasmodically drawn towards the paralyzed side, and its appearance may deceive the careless observer, as in the early period of Bell's palsy the face is of course drawn away from the paralyzed side. These secondary contractures and spasms have been noted by me as occurring in varying degree in a number of cases of facial paralysis. Hitzig refers the symptoms to an abnormal excitability of the oblongata, which becomes developed in an unknown manner in conse-

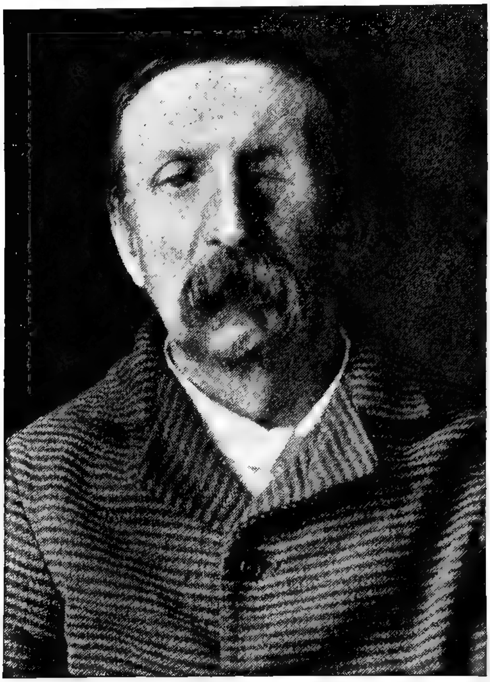
quence of peripheral facial paralysis. I believe with Erb that the condition is not one of "electrical muscle tetanus" induced by electrical treatment, as it occurs in cases in which no electric treatment has been employed. Transient states of spasm in cases of this kind are by no means uncommon. It is generally held that, the facial nerve being purely motor, no sensory or vasomotor disturbances occur in facial paralysis, although the existence of gustatory and secretory symptoms is universally admitted. Frankl-Hochwart, from a study of twenty cases in Nothnagel's clinic, found five cases of sensory, two of vasomotor, and three of combined sensory and vasomotor disturbances. The sensory disturbance was slight in degree, and did not always involve the mucous membrane. He found that these disturbances usually disappeared much sooner than the motor trouble. Reduction of sensation was present after several years in only one inveterate case. Frankl-Hochwart believes that these disorders show that the facial nerve of man contains some sensory and vasomotor fibres, as does the same nerve in animals. Goldzieher observed in two cases of facial paralysis that the eye of the affected side remained dry during weeping, and Jendrássik from observations on four other cases of facial paralysis obtained the same results. Both observers incline to the opinion that the lachrymal gland is supplied by the facial nerve, and not by the fifth as is usually taught. Jendrássik has attempted to work out the exact method of the supply. He believes that fibres are given off from the trunk of the facial nerve as high up as the geniculate ganglion ; these fibres run along the great superficial petrosal nerve to the sphenopalatine ganglion. One group of fibres is distributed to the soft palate. whilst another unites with the second branch of the trigeminus and another runs into the orbital nerve. The orbital nerve unites with the lachrymal nerve, and the combined nerves give off filaments to the lachrymal gland. As peripheral facial paralysis usually comes under observation early, facial atrophy is not, as a rule, a marked phenomenon ; in fact, it would seem to occur late, even in old cases, probably because in most instances some fibres remain undestroyed. Notable wasting in cases of very long standing has, however, been recorded, as in one by Bernhardt. The patient was a man twenty-four years old when studied. A paralysis of the facial muscles of the right side was first noticed two weeks after a natural birth. Most of the muscles of the right side supplied by the seventh nerve were atrophic, and did not respond to faradic or galvanic excitation. The two orbicular muscles, namely, the orbicularis oris and the orbicularis palpebrarum, usually regarded as supplied solely by the facial nerve, seem sometimes to escape partially, or are relatively much less affected than the other facial muscles. Mann, for instance, found that in two cases undoubtedly peripheral the orbicularis oris escaped, although all the other muscles supplied by the facial were completely paralyzed.

The same observer had noted, what has also been recorded by a number of others, that the orbicularis palpebrarum was not so seriously affected as the other muscles in peripheral cases. It is well known that it largely escapes in cerebral cases. Even when the orbicularis palpebrarum is markedly affected it shows a tendency to recover earlier than the other muscles, and one or two cases have been reported in which the orbicularis oris has also recovered its functions before the other facial muscles. Some hold that the frontales as well as the orbicularis palpebrarum are less severely affected than the other parts of the musculature; but this is doubtful. It is not true, as suggested by Gowers, that preservation of the function of the orbicularis oris in facial paralysis always indicates that the disease is cerebral. The position of the tongue in peripheral facial paralysis is a matter of some interest. In some cases it is protruded in a straight line, in others it deviates to the paralyzed side. Hitzig states that in the lighter forms the tongue is always put out straight, but that in severe and protracted cases it deviates to the sound side, if at all. If it does, and the angle of the mouth on the paralyzed side be drawn to its proper place, the tongue will straighten. His explanation is that the tongue is accustomed to keep at an equal distance from each oral angle, and when it finds that it is nearer one angle it deviates to the other side until its median position is restored. Paralysis of the soft palate, in the light of the most recent investigations, is probably a disease of the vagus nerve, or of its nucleus (*nucleus ambiguus*).

Association of Facial Paralysis with other Cranial Nerve Affections. In very rare instances an extension of facial paralysis from one side of the face to the other has been noted. Occasionally one or more branches of the trigeminal nerve may be simultaneously involved with the facial nerve in a paralytic affection. It is supposed that in some of these cases at least this association of disease of the two nerves is the result of spread of inflammation by contiguity of tissues. Cases of herpes zoster facialis and herpes zoster cervicalis occurring with facial paralysis show one of the methods of combination of seventh and fifth nerve disease. The illustration (Fig. 420) shows facial and trigeminal paralysis combined in the same case, the lesion probably being nuclear. This patient was a man fifty-four years of age, with an uncertain history of syphilis, but with a certain record of drinking and of rheumatism. Between March, 1884, and January, 1888, he had three apoplectiform attacks causing more or less paralysis in the left half of the body. In the last, which occurred in January, 1888, he was left with complete facial palsy on the left side. It is uncertain whether at this time or later the affection of the motor branches of the fifth nerve from which he also suffered was developed. Some time in 1889 his left eye was removed by Dr. de Schweinitz because of a trophoinflammatory affec-

tion. The illustration shows great wasting and marked deformity of the entire left side of the face. The appearance is strikingly different from that of the case of facial paralysis shown in Fig. 419, although the same side is paralyzed in both instances. The face in the case shown in Fig. 420 appears to be, and is in reality, smaller upon the left side, and the nose is slightly twisted to the left. A distinct hollow is seen in the position of the left temporal muscle, and the face is much flattened over the position of the left masseter.

FIG. 420.



Facial and trigeminal paralysis, with atrophy.

Etiology.—The etiology of facial paralysis varies according to the focal variety of the affection. Chief among the causes of peripheral facial paralysis (Bell's palsy), with which we have been mainly concerned in the present section, are exposure to cold and draughts, extension of inflammation or suppuration from disease of the ear, syphilitic disease at the base of the brain, and traumatism. By far the largest number of cases occur from exposure. The patient, usually somewhat run down in general health, exposes one side of the face before an open window or door, or in a vehicle, or on a bicycle, or in some similar way is subjected to severe local draught, and at once, or soon after, finds the face paralyzed. In this way a neuritis or perineuritis is probably set up in the branches and extracranial portion of the trunk of the nerve, the inflammation usually extending for a little distance into the aqueduct. The nerve is probably particularly affected in the space formed by the widening of the Fallopian conduit. Occasionally facial paralysis comes on in a patient with rheumatic symptoms in other parts of the body and without a history of special exposure; in other words, the rheumatic poison attacks the facial nerve at its origin or somewhere in its course and causes a neuritis as it assaults nerves and other structures in various parts of the body. The designation "rheumatic" is sometimes applied to the cases which result directly from chilling or catching

cold. This may or may not be a proper method of description. Undoubtedly in some patients with a rheumatic predisposition facial neuritis, like other forms of neuritis, is more likely to originate from slight exposure. Syphilitic disease of the vessels, membranes, or temporal bone at the base of the brain is a not infrequent cause of facial paralysis, and its clinical peculiarities have been spoken of when discussing the peripheral lesions of the intracranial portion of the nerve. The disease is most frequently a gummatous meningitis, although either tumor or true neuritis may of course be the exact specific lesion. Diseases of the ear, especially suppurative otitis media, are universally classed among the most important causes of facial paralysis, and are causes of special importance in children. The trunk of the facial nerve is separated from the tympanic cavity only by a thin partition of bone, and this may be readily worn through by carious or destructive inflammatory processes. Great differences of opinion exist as to the importance of otitis media in causing facial paralysis. Statistics are of little value in deciding a question of this kind: one observer, for instance, reported three cases of facial paralysis out of four hundred and eleven cases of otitis; another did not have a single case of such palsy among fifty cases of otitis; and still another saw only eight cases out of six hundred and eighty-six cases of otitis. (Bernhardt.) In rare instances local treatment of the ear has been followed by a facial neuritis and paralysis. Fractures of the floor of the skull, direct injuries to the ear, surgical operations in the neighborhood of the parotid gland, blows and cuts in the face, and pressure by forceps are among the causative traumatisms. Cases of paralysis of the facial nerve following the extraction of teeth have been observed by Frankl-Hochwart and others. A few cases have been seen in the course of diabetes. Heredity undoubtedly plays a part as a predisposing cause. It occurs more frequently in males than in females, and the majority of those who suffer are between the ages of twenty and fifty years, although the affection may, of course, be present at any age. The palsy attacks both sides of the face in nearly equal proportion, although some statistics seem to show that the right side is somewhat more frequently the site of the disease than the left. In extremely rare cases, hemorrhage into the sheath of the nerve or around the nerve trunk has been recorded. Gowers suggests that some of the cases of abrupt origin and complete type are due to hemorrhage within the Fallopian aqueduct, and he believes that thrombosis in a vessel of a nerve is a conceivable mechanism, especially where the condition of the patient favors the occurrence of thrombosis, as when facial paralysis occurs soon after childbirth. Nuclear facial paralysis is usually due to slow degenerative processes, although focal lesions sometimes attack both nuclei and root fibres of the nerve. Gliomatosis of the bulb is a disease which may attack the facial nuclei and radicles, as it does those of the other bulbar

nerves. Pontile, subcortical, and cortical facial paralysis is usually dependent upon a focal lesion, such as a tumor, hemorrhage, or focus of softening. Double facial paralysis is due to degeneration of the nuclei of both sides, to basal meningitis which spreads from one side of the brain to the other, to a double neuritis attacking conjointly or successively the nerve trunk or branches, and, when supranuclear, to multiple lesions, as tumors, abscesses, hemorrhages, or embolisms. In rare instances supranuclear bilateral facial paralysis has followed injuries to the head which have caused fractures or hemorrhages on both sides of the head or fracture on one side and hemorrhage on the other. Hysterical facial paralysis usually occurs with other stigmata of hysteria, and is commonly partial. Hemiparesis of one side of the face with hemianesthesia of the same side of the body and other hysterical stigmata are likely to be present.

Pathological Anatomy.—Autopsies and microscopical examination in a few cases of peripheral facial paralysis of the complete type have been put on record. In one by Minkowski the patient had an attack of typical complete rheumatic facial paralysis, but committed suicide eight weeks after the beginning of the trouble. Minkowski found a true degenerative neuritis, with disintegration of the medullary sheaths, but without evidences of interstitial inflammation. An important practical feature in this case was the intensity of the inflammation at the outer end of the Fallopian aqueduct in the peripheral distribution of the nerve, this intensity decreasing as the nerve was traced backward to the geniculate ganglion. The central portion of the nerve to the geniculate ganglion and including it was normal. Others have also found a parenchymatous neuritis extending peripherally from the site of the lesion without any interstitial change in the nerve. The pathological evidence does not permit us to say that in the rheumatic form of facial palsy the nerve is compressed by inflammatory material either in, or at its exit from, the Fallopian aqueduct. This evidence is very meagre, and locates the change chiefly in the nerve branches distributed over the face and in the outer part of the Fallopian aqueduct; while the state of the nerve is a parenchymatous and not an interstitial inflammation. (Turner.) With regard to various aspects of the pathology of facial paralysis, there are to be noted, as stated by Turner, the forms associated "(*a*) with chronic degenerative atrophy of the cells of the facial nucleus; (*b*) with implication of the nerve roots by lesions situated in the tegmentum pontis; (*c*) with destruction of the nerve roots by gummatous or other new growths involving the dura of the base of the skull; (*d*) with the form associated with other peripheral palsies, as sequelæ of diphtheria, in which no pathological change at all has been detected; and, lastly, the form which is observed in the true myopathies."

Diagnosis.—The diagnosis of the existence of a facial paralysis

is, of course, readily made. The affection could not be confounded with any other, except by the most careless observation. The points of distinction from a paralysis in the distribution of the motor portion of the trigeminus have been indicated when discussing the symptomatology of this disease, as have also those of hemifacial atrophy. The most important diagnostic points usually are to distinguish between a cerebral and a peripheral facial paralysis, and, with regard to the latter, to have some clear idea as to the site and extensions of the lesion. Most of these differential points have been indicated in discussing the symptomatology of the different focal varieties of facial paralysis. A practical distinction, not always to be depended upon, between a peripheral and a cerebral paralysis is the completeness of the paralysis in the former type. In cerebral cases the remaining palsy is nearly always of the muscles supplied by the branches of the nerve to the lower part of the face. The frontal, corrugator, and orbicular palpebral muscles usually recover so fully that the paralysis attracts little or no attention, although, as has been stated, a minute study of the movements of the muscles will demonstrate some paresis. The electrical examination of a case of facial paralysis will settle the diagnosis of its peripheral nature. Most peripheral cases give typical reactions of degeneration, all the changes, modal and serial, sooner or later being present. In very light forms of the disease, dependent probably upon mild neuritis, the reactions to both the faradic and the galvanic current remain nearly or quite normal. Even partial degeneration reaction may be present in a type of the disease of medium severity, but this is rare, and usually the presence of partial degeneration reaction points to nuclear disease. Normal electrical responses indicate either a cerebral lesion or one in the pons above the decussation of the nerve.

Prognosis.—The prognosis of peripheral facial paralysis is variable. Probably the majority of cases make almost complete recoveries. In a considerable percentage, however, recovery is only approximately complete, and in a few no improvement whatever takes place. In those dependent upon an inflammation attacking the nerve trunk the prognosis as to the amount of recovery is, of course, dependent upon the degree of destruction and degeneration; and the same remark is applicable to cases which occur in the course of suppurative and focal affections, like tumor and hemorrhage. Nuclear focal paralyses, like all other nuclear diseases, are of grave prognosis. Syphilitic cases are relatively favorable. When the palsy is associated with middle ear disease the prognosis is best stated as uncertain. When the secondary spasm and contractures of Hitzig set in, the prognosis as to complete recovery is unfavorable, although even in these cases much improvement may take place. In the earlier stages of the common variety of facial palsy due to peripheral neuritis it is difficult to give an exact prognosis. The

physician should be upon his guard, and, if possible, postpone a positive opinion for two or three weeks, although in a general way a favorable prognosis can usually be given. Electricity is of value in deciding the final outcome of such cases. "If from a week to ten days after the onset of the paralysis there is no quantitative diminution, but rather an increase, to faradic stimulation, the statement may be made that recovery will take place in three or four weeks. If at about the same time after the onset the faradic irritability is distinctly quantitatively lessened, recovery is likely to take place in from six weeks to two months. Should the faradic excitability be entirely lost one week to ten days after the onset, recovery is likely to be delayed for some months. In this case, in addition to the loss of faradic reaction, there is likely to be qualitative galvanic alteration, causing the reaction of degeneration. It is improbable that in such cases complete recovery will take place. If the faradic excitability remains in abeyance three, four, or more months after the onset, the prognosis as to recovery should be guardedly given. In such cases the onset of secondary contracture and overaction is highly probable." (Turner.)

Treatment.—The treatment of facial paralysis must in the first place depend upon its etiology. If the cause is rheumatic or syphilitic, remedies directed to this cause should be employed. In most cases of the variety due to exposure early treatment with calomel and with moderate doses of salicylates and bromides will be found useful; these can be assisted in their action by the use of diaphoretics and baths. A few leeches to the mastoid or below the ear are serviceable in the acute stage. Counterirritants in the same neighborhood may also be used. Turner, however, calls attention to the fact that in the more severe forms of facial paralysis, especially those associated at the commencement with considerable pain, care ought to be exercised in the use of counterirritants. He records a case in which the injudicious use of croton oil provoked intense cellulitis over the parotid region, and he believes that in such cases the careful use of small mustard leaves in front of the ears, repeated several times, may be of greater service than the application of a blister. My own experience is in accord with that of this observer. When disease of the ear is present, this should be treated according to the rules and methods applicable to the treatment of such diseases, independently of the existence of the paralysis. When the disease tends to become chronic, the iodides may be given in moderate doses, and occasionally the administration of large doses of iodide for a short time seems to start the nerve on the way to recovery. Both strychnine and electricity may be used after the acute stage has subsided, but, as a rule, never before ten days or two weeks have elapsed. In using electricity for any form of facial paralysis the weakest current that will cause a visible contraction is sufficient; either anode

or cathode may be used to the muscle or nerve periphery, but the best plan is at first to use the anode to the nape of the neck or under the ear, and the cathode to various points in the distribution of the muscles and nerve branches. It is best to begin with two or three milliamperes of current strength, and to increase until a visible contraction is produced. Great care should be taken in applying the electrodes or removing them not to do so abruptly. The resistance should not be increased or cells introduced without consideration, as often a current previously weak or imperceptible will suddenly appear in full force and harm may be done in this way to the retina or by producing vertigo. When no response can be obtained with a fair number of cells and an amount of resistance which should give a decided current, the electrodes and cords should be carefully examined. The lack of current may be dependent upon some temporary disturbance in the battery. One of the electrodes can be used inside of the mouth with advantage in some cases. A clean electrode is of course essential, as for instance one freshly covered with absorbent cotton. The electrode placed at the nape of the neck or under the ear should be larger than that used for treating the face. The cathode should be a motor point electrode, or at least one small enough for the individual muscle to be treated with it. The treatment should not be too long: five minutes will commonly be sufficient: much harm can be done by overtreating. In the early period of electrical treatment the paralyzed muscle may respond and contract with a weaker current than is required by the muscles on the unaffected side, and the normal response, even in mild cases, is often changed, so that the contraction produced by closing the circuit at the anode or positive pole will exceed that produced by doing the same at the cathode. As the muscles begin to recover, the response to galvanism begins to diminish and that to faradism to return, hardly noticeable at first, but gradually increasing. If there is any tendency to contracture, all electrical treatment is temporarily contraindicated, for although this is not the cause of the contracture it may tend to increase it. It is a mistake to go on using faradism week after week, as is sometimes done by general practitioners, in a case of facial paralysis in which the muscles refuse entirely to respond to this current. A strong faradic current will of course produce contraction in the masseter or other muscles supplied by the fifth nerve, which might mislead a very careless observer.

FACIAL SPASM.

Definition and Synonyms.—By *facial* spasm is meant spasm implicating the muscles supplied by the seventh or facial nerve. In the sense in which it is used here, facial spasm does not include spasm attacking the muscles of the motor distribution of the trigeminus or the ocular muscles. In its most common form it usually combines both

clonic and tonic movements. It has various synonyms, as *painless tic, histrionic spasm, mimic* or *mimetic spasm,* and *convulsive tic.* Besides the forms which go by these names, other special forms of facial spasm are met with, as, for instance, the secondary contractures of Hitzig described in the last section; the facial spasms of diplegia, particularly of the type known as bilateral chorea and double athetosis; forms of hysterical spasm; and forms which appear to be of the nature of obsessions or monomanias, the patients having the habit of twitching some part of the face, the habit often growing and becoming permanently implanted upon the nervous system.

Symptomatology.—*Common Type of Painless Tic.* In the majority of cases the disease comes on gradually, at first perhaps affecting only one or two muscles or sets of muscles. Probably at the beginning the muscles most frequently affected are either the orbicularis palpebrarum or the elevators of the angle of the mouth. The spasm is, as a rule, unilateral at first, and it may continue throughout to be unilateral, or may become bilateral only after a number of years. In some cases, however, even when chiefly unilateral, certain of the muscles on both sides of the face may be attacked with spasm at the same time, as for instance the corrugator supercilii, the orbiculares of the eyelids, and the frontal muscles, when, so far as the rest of the face is concerned, the spasm is confined to the muscles on one side. In other words, those muscles on both sides of the median line which most frequently act together physiologically, and which are probably nearly equally innervated from each side of the brain, are those which are most likely to be affected with bilateral spasm. In not a few cases, after many years, all portions of both sides of the face become involved, although they are rarely equally involved. The side first affected is usually the one which to the last is most affected. In the most common type of facial spasm, a form which has been termed idiopathic, the spasm attacks only one or two of the muscles at first, and those at infrequent intervals. Perhaps the patient or his friends observe a slight twitching of the orbicularis palpebrarum, of the levator anguli oris, or of the zygomatics. These twitchings become more pronounced, occur more frequently, and spread to neighboring groups of facial muscles. After an indefinite time a marked admixture of tonic and clonic spasm is usually observed, the attack beginning with twitchings, but during its course some of the muscles becoming strongly contractured. The clonic spasm is at times imposed upon the tonic spasm, the muscles of the face, for instance, being drawn to one side, and the eye being spasmodically closed, while at the same time slight or severe jerking movements affect the muscles at intervals. The spasm is usually strictly confined to the distribution of the portio dura, but occasionally the muscles supplied by other nerves may be affected. If the muscles of mastication, or the stylohyoid, mylohyoid, and digas-

tric muscles, or the velum palati, are affected along with the facial, this indicates that the case is not one of the usual type of mimic spasm, but is due to some widely distributed organic lesion, affecting also the trigeminal, vagus, and perhaps other nerves. Sometimes the muscles on the paralyzed side gradually grow atrophic, but the process of wasting is usually so extremely slow that it can scarcely be determined until the disease is of many years' standing. In some cases this atrophy does not seem to take place, and even hypertrophy may result from the constant overaction of the spasmodically affected muscles. With regard to atrophy, I have seen at least three types: (1) cases in which the muscles remain for a long time, if not altogether, unchanged; (2) cases in which the muscles waste with exceeding slowness; and (3) cases in which a comparatively rapid wasting takes place. While there is usually no marked paralysis in cases of facial spasm, yet occasionally some paresis is present, but does not attract attention. The electrical reactions either are not changed or are those of partial degeneration. Vasomotor changes are not, as a rule, present. The spasms come on under excitement or after fatigue, and they can be induced sometimes by mechanical irritation of the muscles, or by having the patients try to use the muscles of the face. Rest and darkness have an influence in diminishing the spasm. The description which has been given applies especially to the common type of slowly progressive and continuous facial spasm, the pathology of which is as yet not clearly known. Cases of other types give special peculiarities, and therefore they should be separately considered.

Cortical and Subcortical Facial Spasm. Several cases of cortical facial spasm have been put on record. One of the earliest and the most remarkable of these is the case recorded by Berkley. In this case the spasm was confined to the zygomatics of the right side of the face. Berkley on autopsy found in the precentral convolution of the right side, about one and a half inches above the margin of the Sylvian fissure, a nodule of calcareous degeneration, nearly circular in shape, and about three sixteenths of an inch in diameter. Its depth was about one half the thickness of the cortical gray matter. A large number of cases of cortical facial monospasm have been reported in recent years, few of them, however, being of pure types. Parts of the body other than the face are generally sooner or later involved. Facial spasm is not infrequently the signal symptom in cortical or subcortical disease. In a case recorded by Seguin and Weir the first symptom noticed was a spasm in the right cheek and neck, the head and face turning to the right. For about three years the twitching spasms affected only the muscles of the face. Later the patient had attacks in the right upper extremity and right lower extremity, and these attacks were accompanied with unconsciousness. His speech gradually became thick. A growth of the shape and size

of the end of the forefinger was found, entirely in the white substance. In this case, as in similar ones, the concomitant symptoms were of value chiefly in the diagnosis of the location of the lesion causing the spasm.

Facial Spasm due to Small Gross Lesion of the Nucleus or Nerve Trunk. Spasm in the muscles of the face—those supplied by the portio dura—may be produced by a small gross lesion anywhere from the facial nucleus to the peripheral distribution of the nerve. Cases have been reported in which tumors, aneurisms, or foci of softening or degeneration have caused this symptom. These lesions have been so situated as to affect the nerve in the substance of the pons, at its superficial origin, or in the auditory meatus. In one case, for instance, an aneurism, in another a tumor of the pons, in another a gummatous meningitis, and in another an abscess of the petrous bone gave rise to facial spasm either with or without accompanying symptoms. In facial spasm due to gross lesion, whether cortical, subcortical, nuclear, or neural, the spasm is likely to be coarse and more irregular in type than in those cases of painless tic which are most frequently seen, and which we are inclined to believe to be due to nuclear instability dependent upon nuclear degeneration.

Blepharospasm. Blepharospasm is occasionally a more or less intractable spasmodic affection. It is caused by a spasm, usually tonic, of the palpebral orbicular muscle. Sometimes a clonic or a clonicotonic form of blepharospasm manifests itself by an unpleasant uncontrollable blinking or winking of the eyelids. One form of clonic blepharospasm is sometimes called *nictitating spasm;* it is, in fact, simply an abnormal winking or blinking. In the worst forms of this affection the eyelid is so tightly closed as to be painful to the patient. If the eyes should be open or partly open, the slightest exposure to light or irritation will cause them to be snapped shut, and so to remain in spite of the voluntary efforts of the patient and even physical efforts to open them. Blepharospasm may be due to various causes, as to peripheral irritation from a foreign body in the eye, an inverted eyelash, a slight conjunctivitis, granular lids, or a phlyctenular ulcer. The instillation of various myotics and mydriatics used by the ophthalmologist sometimes causes blepharospasm. Eserine, for instance, may give rise to clonic fibrillary contractions of the eyelids, and cocaine sometimes produces a tendency to spasm of the levator palpebræ. Sometimes no direct tangible cause of the blepharospasm is to be found in the condition of the eye or its coverings, the most intractable cases being of this type: thus it may be from irritation of the ophthalmic branch of the fifth nerve, or from irritation reflected from a tooth or from some other portion of the face or head. Other cases have been attributed to ulcers in the mouth and nose, or to irritation of the pharynx. The so-called functional or hysterical blepharospasms often obstinately resist treatment. On

the other hand, they may be temporarily relieved by pressure upon the branches of the fifth nerve which go to the orbit, or upon the branches of the facial which go to the frontalis, corrugator supercilii, or levator palpebræ. In very rare instances blepharospasm may be of central and even of cortical origin, due to irritation of that portion of the facial centre in the cortex which is concerned especially with the orbicular movements. As bilateral consentaneous movements are represented in each side of the brain, irritation of one hemisphere might in rare instances give rise to double blepharospasm. A lesion would, however, have to be very small indeed so to affect the cortex or subcortex as to cause spasmodic closure of the eyelids without affecting other parts of the face. As blepharospasm has distinctive clinical features, its diagnosis, prognosis, and treatment will be here briefly considered. The diagnosis is to be made by a careful inspection of the patient. It could be confounded only with a paralytic or spasmodic ptosis. The prognosis is variable. Cases due to peripheral irritation, to the instillation of drugs, to unequal exposure to light, or to similar removable causes, usually make speedy recoveries. Some cases obstinately resist all medicinal and mental therapeutics, and for these surgery is the only resource. The surgical operations which have proved most beneficial are such as stretching or resection of branches of the fifth nerve and stretching of the branches of the facial to the orbicularis palpebrarum. Some of the cases of blepharospasm are due to refraction errors and the resulting irritation of the conjunctiva. Children sometimes suffer from a persistent form of cramp of the eyelids for which no known cause can be discovered.

Etiology.—In discussing the etiology of facial spasm it is important to separate cases of the most common type from others, as they constitute a distinct clinical entity. In this class the etiology is obscure. I have seen at least a score of such cases, and yet have never been able to come to any satisfactory conclusion as to their etiology or pathology. Not infrequently they occur in patients with a neuropathic heredity, although I know of only one instance of apparently direct transmission, and that may have been a coincidence. This is a case referred to by Gowers of a mother and daughter who were both affected with facial spasm late in life. Of course not a few cases of spasm involving the face or other portions of the body have been recorded as occurring in different members of the same family, but these are not instances of true painless tic. The disease usually comes on after middle life, most frequently between the ages of fifty and sixty, although it may be observed in rare instances at almost any age. It is more common in women than in men. Exhausting diseases, emotion, great fatigue, and injuries are assigned as exciting causes. It has been observed during pregnancy, and cold and exposure in some instances have seemed to be factors.

The reflex variety of facial spasm is most frequently due to trigeminal irritation, as when it arises from a decayed tooth or from injury to a nerve anywhere in the face or head. The causes of the cortical, subcortical, pontile, and basal forms of facial spasm have been sufficiently discussed when treating of these varieties of spasm. The causes are, in brief, irritative focal lesions of all sorts, as tumors, abscesses, localized meningitis, aneurism, embolism with softening, focal hemorrhages, exostoses, etc.

Pathology and Morbid Anatomy.—So far as known to me, no case of painless tic of the ordinary type with autopsy and microscopical examination has been put on record. The disease would seem to be due to some affection of the facial nucleus, or possibly of the cortical facial centre. The ganglionic disease is most probably of a degenerative and progressive type. Exceedingly slow degeneration of the facial nucleus would seem to account best for some of the cases; in others, in which atrophy does not take place even late in the affection, degeneration of the cortical centre for the face would seem more probable. As the aggregation of nerve cells which constitute a "centre" decreases, the centre itself, regarded as a whole, becomes less stable and less uniform in its method of action. The facial nucleus in the oblongata, for instance, if more or less degenerated, will transmit the current of cortical impulses with less and less smoothness, and spasm instead of uniform physiological action will result. As already indicated, some forms of facial spasm are due to small gross lesions, or are reflex, hysterical, or of habit causation.

Diagnosis.—The diagnosis of facial spasm is of course to be made by the fact of the existence of spasm in a few or in all of the muscles supplied by the seventh nerve. It is necessary first to determine that the case is one of the progressive degenerative type, and this diagnosis is chiefly made by a history of the slow, insidious progress, by the concurrence of clonic and of tonic spasm in the same attacks, by the tendency to diffusion over part of the face, and by the obstinate resistance of the spasm to all methods of treatment. This and some other forms of facial spasm must be distinguished from the secondary spasms and contractures which follow Bell's palsy. It is most important to look for a previous history of true peripheral facial paralysis. When facial spasm is hysterical, other evidences of hysteria are usually present; when it is a habit chorea, the history of the case will be the chief assistance. Hemifacial atrophy, which is a disease of the fifth and not of the seventh nerve, must not be confounded with a case of facial spasm or paralysis in which some atrophy has taken place. It is not impossible to have either facial spasm or facial paralysis associated with true hemifacial atrophy, but the combination is so exceedingly rare as to make it scarcely worth while to take it into account.

Prognosis.—The prognosis in the ordinary type of facial spasm

is always serious. Although many cases of the disease of this type have fallen under my observation, either directly or in consultation with others, I have never seen or known of a case of recovery. To my mind this form of facial spasm is simply one of the forms of degenerative disease of which we have so many examples. Cases due to reflex irritation may recover when the source of irritation is removed. The removal of carious teeth may give relief, although when cases have apparently been of reflex origin the removal of the exciting cause does not always cure the affection. This is probably due to the fact that a spasm habit has been set up.

Treatment.—Treatment of facial spasm is, as a rule, unsatisfactory. If due to a cortical lesion, as to a tumor, spicule of bone, or other irritative lesion, this may possibly be removed. Excision of the cortical facial centre has been practised with reported benefit. In Weir and Seguin's case the removal of a subcortical sarcoma gave relief for a considerable time. Mercury and iodides should be used where syphilis is suspected, or even in other cases of focal lesion of unknown origin, as these absorbents have some efficiency in nonsyphilitic cases. The treatment of the progressive form is unsatisfactory, although relief is sometimes obtained. The medical measures resorted to are both local and internal. Of local remedies the most important are cold, heat, and electricity. Mitchell has strongly recommended the use of local cold, as freezing the cheek with ice or with rhigolene or chloride of ethyl spray. Very hot applications and strong counterirritation over the place of emergence of the nerve from the stylomastoid foramen may be tried. The actual cautery may also be applied in this position, or at any point or points along the course of the nerve. The only electrical method worthy of trial is the use of a weak galvanic current, applied after the stabile method. The anode can be applied under the ear or at various points over the distribution of the nerve, and the cathode at some indifferent place, as at the back of the neck or the sternum. This is the unipolar method, and offers some hope of relief. Morphine, or atropine, or both combined, may be administered hypodermatically. Internally the most useful remedies are the metallic tonics, such as chloride of gold and sodium, oxide or nitrate of silver, and the salts of zinc, copper, and arsenic. Strychnine in increasing doses has been used with reported success. Two of the most valuable narcotic sedative drugs for this as for any other form of spasm are conium and gelsemium. Coniine hydrobromate can be administered in doses of from one fiftieth up to one tenth of a grain three or four times daily. Gelsemium is best used in the form of a fluid extract, beginning with four or five minims and increasing until the patient takes from ten to fifteen or more. Stretching of the nerve has been employed, the immediate result being facial paralysis with cessation of the spasm, but in most instances the spasm returns.

SMALL GROSS LESIONS OF THE PONS AND PREOBLONGATA.

Structures of the Pons and Preoblongata.—In this and the two preceding chapters the diseases of the cranial nerves from the third to the eighth inclusive have been considered. These nerves either arise, or in large part take their intraencephalic course to reach the surface of the brain, in those subdivisions of the encephalon which, in accordance with the terminology adopted in this work, are designated as the pons and the preoblongata. (Fig. 421.) So many structures are compacted into the small space included within the pons and preoblongata that small gross lesions affecting them must affect various important structures besides the cranial nerves, and in this way give rise to special syndromes. It will facilitate the focal diagnosis of lesions in this region to have some readily applied topographical scheme by which the most frequent combinations of symptoms can be understood at a glance. A series of semidiagrammatic illustrations which will be found useful in the study of the focal diagnosis of lesions of the pons and preoblongata have therefore been prepared. In these illustrations the combined pons and preoblongata are represented as divided into thirds in two directions, so that the attention of the diagnostician can be focussed upon any one of nine segments. They can be used both as supplementary and as complementary to the facts already mentioned under the symptomatology and diagnosis of the cranial nerves to which allusion has been made.

FIG. 421.

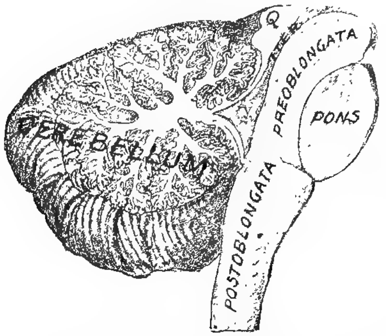


Diagram showing the relations of the pons, preoblongata, and postoblongata to the fourth ventricle, iter, quadrigeminum, and cerebellum: *Q*, quadrigeminum; *V*, fourth ventricle.

Ventral, Lateral, and Dorsal Views of the Pons and Preoblongata.—Ventral, lateral, and dorsal views of the appearances presented by the pons and preoblongata are given in Figs. 422, 423, and 424. These are represented as divided into thirds by horizontal dotted lines which on the different surfaces correspond with one another as determined by sections of actual specimens, the planes of the sections being at right angles with the cerebrospinal axis. The third nerves (*III*) are seen emerging between the cephalic third and the crus, and at the junction of the pons and the postoblongata are the abducens (*VI*) and more laterally the facial (*VII*) and eighth (*VIII*) nerves. On the lateral aspect of the cephalic third, near the crus, the trunk of the fourth nerve (*IV*) appears, and the fifth nerve (*V*) in the middle third, while the seventh and eighth nerves are

FIG. 422.

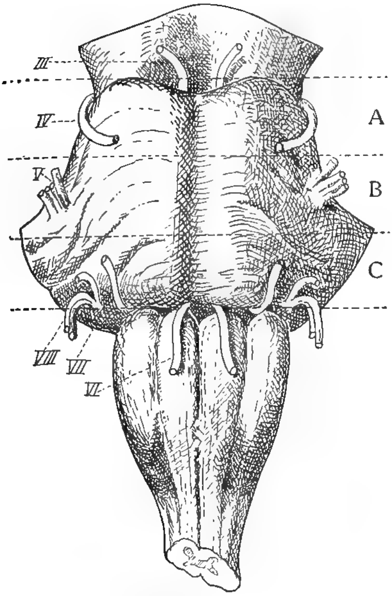


FIG. 423.

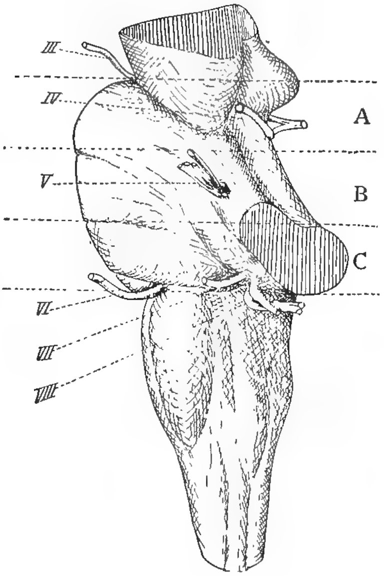


FIG. 422.—Subdivision of the ventral aspect of the pons into thirds, showing the relations of important structures to each subdivision.

FIG. 423.—Subdivisions of the lateral aspect of the pons and preoblongata. The nerves in this and the preceding illustration are indicated by Roman numerals.

FIG. 424.

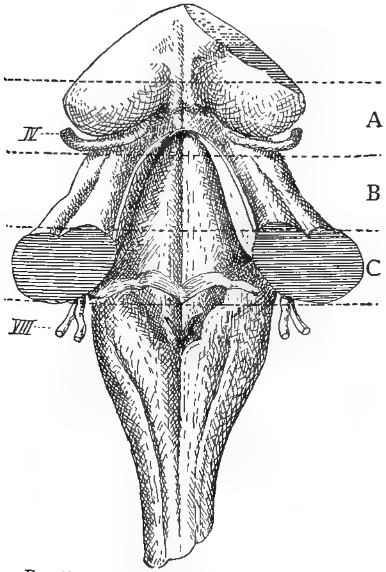


FIG. 425.

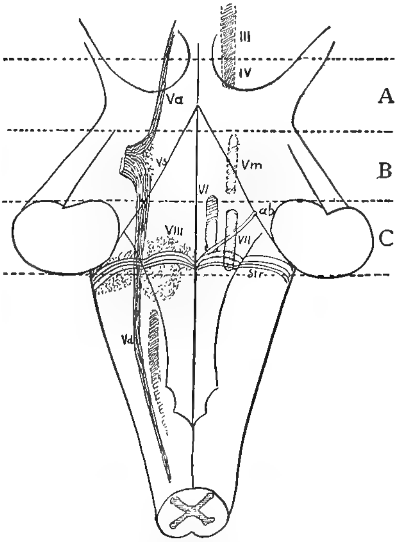


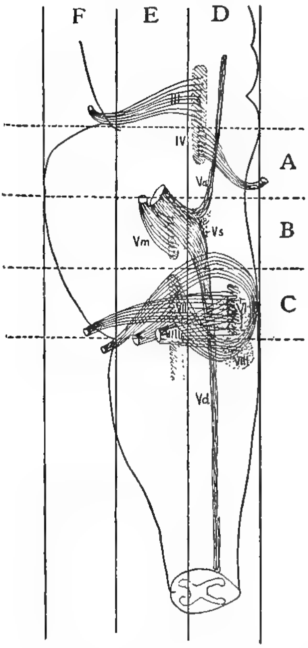
FIG. 424.—Subdivisions of the dorsal aspect of the preoblongata, showing the surface structures. The nuclei and nerves in this and the next figure are indicated by Roman numerals.

FIG. 425.—Subdivisions of the dorsal aspect of the pons and preoblongata, showing the position of the nuclei and root fibres of the cranial nerves from the fifth to the eighth. Va, descending (mesencephalic) root of the fifth; Vs, sensory nucleus of the fifth; Vm, motor nucleus of the fifth; Vd, descending (spinal) root of the fifth; Str, acoustic striæ.

seen in the lower margin of the lateral as well as of the ventral third. In the cephalic division (A) of the dorsal aspect (Fig. 424) are seen the postgeminum or posterior tubercles of the quadrigeminal body, and the fourth or pathetic nerve at its origin and in part of its course. The middle third (B) includes the prepeduncle and a portion of the floor of the fourth ventricle. Both the middle and caudal thirds are partly traversed by the medipeduncle, and the caudal third (C) by the acoustic striæ.

Relative Positions of the Nerve Nuclei and Course of the Root Fibres.—With the aid of Fig. 425 the relative positions of the nuclei of the third, fifth, sixth, seventh, and eighth nerves are made evident. The nuclei of the third nerve are situated in the gray matter along the course of the Sylvian aqueduct cephalad of subdivision A, and the nucleus of the fourth nerve adjoins the nuclei of the third in the upper portion of this cephalic segment, lying chiefly beneath the postgeminum. Both the sensory and motor nuclei of the fifth nerve are in the middle third, while the nuclei of the sixth and seventh and a large part of the eighth are in the caudal or lower third. Hence, when nuclear symptoms referable to any one or more of these nerves are present, the topography of the lesions causing them can be understood and readily stated with reference to these subdivisions. The root fibres of the nerves which spring from these nuclei have, it must be remembered, within the substance of the preoblongata and pons, courses of considerable extent and complexity before they appear on the ventral and the lateral aspect of the pons. In Fig. 426 the dorsoventral courses pursued by the root fibres of the third, fourth, fifth, sixth, seventh, and eighth nerves are approximately indicated. In this illustration lines are shown, drawn longitudinally and at right angles to those already used in making the subdivisions A, B, and C, thus giving in lateral view nine segments, the thirds A, B, and C each being subdivided into thirds,—three ventral, three dorsal, and three intermediate (that is, intermediate between the dorsal and the ventral segments). The root fibres as well as the nuclei of the third nerve chiefly lie cephalad of all the

FIG. 426.



A lateral diagrammatic view of the course of the root fibres of the cranial nerves from the third to the eighth. The diagram is shown subdivided into thirds longitudinally (D, E, and F) as well as horizontally (A, B, and C).

above subdivisions. In the cephalodorsal segment (A) are seen the root fibres of the fourth nerve and the descending (mesencephalic) root of the fifth. In the dorsal and intermediate segments of the middle third (B) both the spinal and the mesencephalic roots of the fifth nerve are seen, as are also a few of the curving roots of the seventh nerve; while in the dorsal and intermediate segments of the caudal subdivision (C) are crowded most of the root systems of the sixth, seventh, and eighth nerves.

Fig. 427.

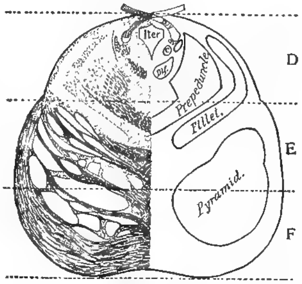


Fig. 428.

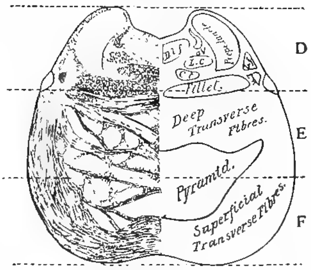
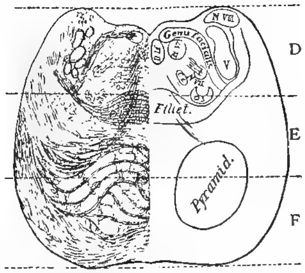


Fig. 429.



Figs. 427, 428, and 429.—Transections of the pons and preoblongata (Fig. 427 through about the middle of the subdivision A, Fig. 428 through B, and Fig. 429 through C, of Figs. 422, 423, 425, and 426). These figures show the relations and position of the most important structures: *O.S.*, superior olive; *C.t.*, central tegmental tract; *L.c.* and *c.*, locus ceruleus; *D.l.f.*, dorsal longitudinal fasciculus; *L.f.*, lateral fillet; *L.n.*, lateral nucleus. The numerals refer to nerve nuclei and roots.

Structures shown in Transections of the Pons and Preoblongata.—Making now dorsoventral sections through the middle of each of the subdivisions A, B, and C, the appearances of such transections, as seen under low powers of the microscope, would be much as shown on the left half of Figs. 427, 428, and 429. Fig. 427 shows the appearance of a section through the middle of the subdivision A, Fig. 428 of one through the middle of the subdivision B, and Fig. 429 of one through the middle of the subdivision C. In these figures are brought into view the appearances in section not only of the root fibres and nuclei of the nerves, but also of various important longitudinal and transverse tracts,—the superficial and deep transverse fibres of the pons, the pyramidal tract, the fillet, the peduncles, some of the most important of the nuclei of the cranial nerves and root fibres, and certain special nuclei and tracts, as the superior olive, the lateral nucleus, the dorsal longitudinal bundle, and the central tegmental tract. The merest glance will show that in all positions the most important parts involved in lesions of the ventral and of the intermediate segments will be the superficial and deep transverse fibres and the pyramids. The fillet lies about the junction of the intermediate and dorsal segments,

varying in its median and lateral positions in different cephalocaudal sections. Nuclei and root fibres are most prominent in dorsal segments, while the pyramidal tract dominates the symptomatology of ventrally situated pontile lesions.

Lesions of the Pyramidal Tract in the Pons.—The pyramidal tract is the great tract from the motor region of the cerebral cortex. Hence destructive lesions of this tract will always give paresis or paralysis of the opposite extremities. When the lesion is in the cephalic portion of the pons, before the decussation of the pyramidal fibres for the trigeminal, abducens, and facial nerves, some ocular and especially some facial paresis may be present. This loss of power in the face must be distinguished from that caused by lesion of the facial root fibres or nuclei. The paralysis of the face is central, and has features similar to that shown in the arm and leg. It is usually incomplete, although it may be more marked than when caused by a lesion situated higher in the pyramidal tract or in the cortex. It does not give the electrical reactions of peripheral facial paralysis. Irritative lesions of the pyramidal tract in the pons and elsewhere may give rise to monospasm, or even to unilateral convulsions, chiefly affecting the limbs and face of the opposite side. Often, in lesions of the ventral portion of the pons, the cranial nerves at, just before, or just after their superficial origins will be involved, giving various forms of so-called alternate hemiplegia.

Functions and Lesions of the Superficial and the Deep Transverse Fibres of the Pons.—The superficial transverse fibres of the pons which lie ventrally to the pyramids contain chiefly cerebellar fasciculi. In the middle third of the pons they run nearly horizontally, having a more oblique course in the lower and upper thirds. They connect special portions of opposite halves of the cerebellum, and also portions of the lateral lobes of the cerebellum with the opposite cerebral hemispheres. The symptoms given by lesions of special bundles of these superficial transverse fibres are not yet thoroughly known. I have reported a case in which, apparently as the result of a small hemorrhage involving these fibres as well as some of the deeper parts of the pons, the patient had atrophy of one lateral lobe of the cerebellum and of the opposite hemicerebrum. Passing to the intermediate segments, as shown in the transections Figs. 427, 428, 429, it will be seen that the pyramidal tract occupies part of this region, which also contains the deep transverse fibres and, near the junction of the intermediate and dorsal thirds, the fillet. A lesion centrally situated in the substance of the pons will, therefore, give symptoms the result of the destruction of these deep transverse fibres, and, if the lesion extends ventrally and dorsally, of both the pyramidal tract and the fillet. The deep transverse fibres constitute largely the structure known as the trapezium, or trapezoid body. In large part, at least, it is the path of

the cochlear nerve. Fibres of the trapezoid body pass to the various auditory nuclei, and also to the roof nucleus of the cerebellum, and connect with the superior olives both of the same and of the opposite side. According to some authorities the trapezoid fibres unite with the fillet, and according to others they intersect it. While the lower or caudal border of the trapezium corresponds nearly to the same border of the pons, cephalad it reaches only as high as the place of emergence of the sensory root of the fifth nerve, and therefore it would be chiefly involved in lesions of the two lower thirds of the pons, as given in the diagrams.

Functions and Lesions of the Lemniscus, or Fillet.—As the fillet, or at least its most mesal portion, is a part of the great sensory tract, its lesions cause disorders of sensation. Numerous cases have been reported in which deeply situated pontile lesions have given rise to anesthesias, and especially to impairment or loss of the senses of pain and temperature; and numerous studies of degeneration following focal lesions have also shown the part played by the lemniscus as a sensory tract. The acoustic tract runs in the lower lateral division of the fillet to the postgeminum, so that lesions of the lateral fillet, like those of the trapezoid body, should cause affections of hearing.

Functions and Lesions of the Superior Olive.—The superior olive, which generally extends the whole length of the pons, is closely approximated to the dorsal surface of the trapezium, and is located, therefore, near the junction of the dorsal and intermediate segments. A function that has been assigned to the superior olive is to act as a reflex centre for correlating the movements of the head and eyes with auditory impressions, and its lesions might therefore be expected to interfere with such correlations, causing a lack of response by the head and eyes to sounds coming from various directions. The connections of the superior olive which have been traced are with both accessory nuclei of the auditory nerve, with one or possibly both cerebellar roof nuclei, with the nucleus of the sixth nerve on the same side, with the dorsal longitudinal fasciculus of the opposite side, with the lateral columns of the cord, and with the postgeminum.

Lesions confined to the Preoblongata.—In the dorsal or preoblongatal portion of the region we are considering the structures are complicated. Here in various positions are the nuclei and root fibres of the fourth, fifth, sixth, seventh, and eighth nerves, and, in addition, certain special fasciculi, as the dorsal longitudinal bundle and the central tegmental tract. Dorsal lesions in different fore-and-aft segments will, therefore, give varying forms of paralysis of the face, of trigeminal paralysis, motor and sensory, and of single or associated ocular palsies. As the third, fourth, and sixth are all nerves to the ocular muscles, and as in various movements of the eye these

muscles act to a greater or less extent together, both on the same side and across the median line, the nuclei of these nerves must be anatomically connected, and lesions of their associating and correlating fibres will give special disorders of ocular movements. Some of the connections, it is believed, are by the dorsal longitudinal bundle. Many of the curving strands in the pons and oblongatas run from the nuclei of the motor cranial nerves, partly to the opposite dorsal longitudinal bundle and partly to this fasciculus on the same side.

Cases illustrating Segmental Localization in the Pons and Preoblongata.—The records of one or two cases may serve to emphasize some of the points just made with regard to localization of small gross lesions in the pons and preoblongata. In one of my cases autopsy showed a lesion the location and limitations of which are indicated in the illustration Figs. 430 and 431. A transection through the pons within the limits of subdivision B of the diagrams just given revealed a ventral lesion near the mesal line in the right half of the pons. This in the fresh specimen appeared to be limited as shown in the drawing Fig. 430. Subsequently microscopical sections were cut and mounted by Dr. A. O. J. Kelly, from which, among other drawings, the one shown in Fig. 431 was made. The microscopical section demonstrated that the area of softening and degeneration extended much farther than appeared in the fresh specimen.

Fig. 430.

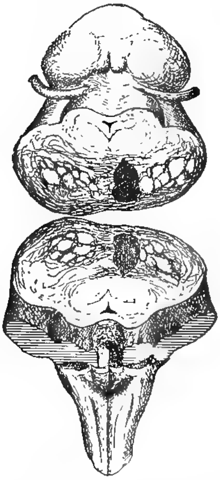


Fig. 431.

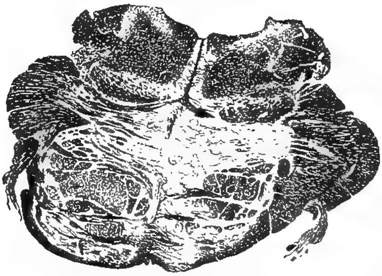


Fig. 430.—Drawing showing the position and apparent size of a pontile lesion in the fresh specimen. The patient had paralysis of the left rectus externus and paresis of the right rectus externus, with restriction of ocular movements, paresis which deepened into paralysis of the left extremities, exalted reflexes, and great emotionality. A photograph of this patient is shown in Fig. 395, p. 827.

Fig. 431.—Microscopical appearances of the lesion in the case shown in Fig. 395 (p. 827). The section was made near the position of the lesion as shown in the fresh specimen. (Fig. 430.)

The destruction and degeneration involved both pyramidal tracts, the deep transverse fibres, the superficial transverse fibres, and the root fibres of the right and left abducens nerves. The destruction was much greater on the right than on the left. A summary of the symptoms shown by the patient is given in the legend to Fig. 430.

In a second case the lesion was a circumscribed softening most extensive near the ventral surface of the pons at its cephalic extremity. It tapered and became more dorsally situated as it extended towards the postoblongata. About the middle of the pons it slightly crossed the mesal line. The lesion did not quite reach the postoblongata. The appearances presented by this specimen in the fresh state are shown in Figs. 433, 434, and 435, and the microscopical appearances in Fig. 436. The different symptoms presented by the patient are summarized in the legend to Fig. 432. Another case of central softening of the pons has also been reported by me. None of the cranial nerves were superficially involved, although the lesion was unusually large. The clinical history showed headache, a vertiginous seizure followed by partial right hemiplegia, left convergent strabismus, defective articulation, and paralysis of the left arm three weeks after the attack. Another seizure was accompanied by excessive emotionality, profuse perspiration, difficulty in breathing and swallowing, inability to speak and to thrust out the tongue or open the mouth wide, and paralysis of both arms and legs; and at last conjugate deviation to the right. In a case of tumor of the pons, the chief symptoms of focal lesion were hemiparesis, partial right ptosis, diminished sen-

FIG. 432.

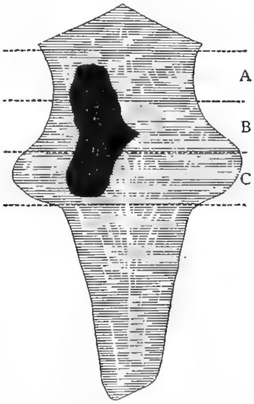


Diagram showing the anteroposterior dimensions of a pontile lesion in a case in which the chief symptoms were right facial paralysis, paresis of the right arm and leg, paralysis of the left internal rectus, paralysis of consentaneous ocular movements to the left, and exalted reflexes. The microscopical appearances of this lesion in transection are shown in Fig. 436.

FIG. 433.

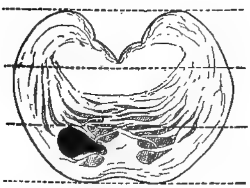


FIG. 434.

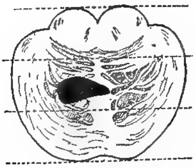
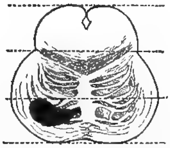


FIG. 435.

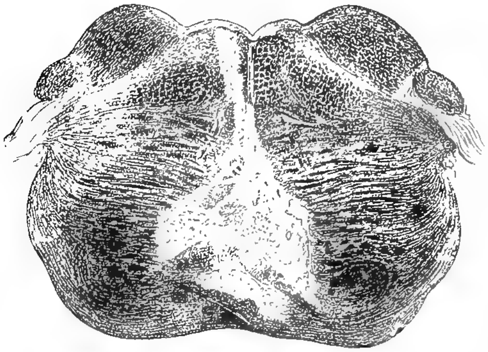


FIGS. 433, 434, and 435.—The appearances in the fresh state of the pontile lesion in three transections (through A, B, and C, in Fig. 432). It will be seen that the lesion as it descends approaches more and more the dorsal aspect of the pons.

sation on the left side of the face and right limbs, conjugate deviation of the eyes and rotation of the head to the right, persistent epistaxis, and a tendency to hemorrhage from the mucous membranes. The autopsy revealed a gumma about half an inch in diameter, distinctly limited to the left cephalic quarter of the pons. Conjugate deviation of the eyes and head is a not infrequent accompaniment of

lesions and especially of tumors towards the cephalic extremity of the pons. In another case of tumor of the pons observed by the author the important symptoms were right external strabismus, with contraction of the right corner of the mouth; persistent cough; staggering; left hemiparesis with left partial hemianesthesia; severe constipation; difficulty of articulation; and late in the history of the case difficulty of swallowing and paralysis of the limbs. On autopsy a large cyst filled with bloody fluid and detritus was found occupying the lower half of the pons and the upper half of the postoblongata, extending a little lower and higher on the right side than on the left. The floor of the fourth ventricle bulged slightly. A man about twenty-six years of age, with a history of syphilis, was admitted to the nervous wards of the Philadelphia Hospital. About two weeks before admission he observed that the right side of his face was paralyzed and insensitive. When admitted, examination showed complete paralysis of the right facial or seventh nerve, also of the muscles supplied by the motor division of the fifth nerve. The patient was also anesthetic on the right side of the face, including the conjunctiva and cornea. The hearing, as determined by the watch, was good on the left side, but on the right it was necessary to bring the watch close against the ear before its tick could be heard. He had, however, no tinnitus. Taste was lost on the right side both on the anterior and on the posterior portion of the tongue. He had neither paralysis nor anesthesia of any part of the body below the neck. The symptoms in this case were probably due to a lesion of the lateral aspect of the pons, although they might be explained by a lesion within the substance of the pons and preoblongata so situated as to destroy the sensory and motor root fibres of the fifth, the facial and glossopharyngeal root fibres, and some of the acoustic root fibres, or perhaps fibres of the auditory tract in the trapezoid body or the lateral fillet. The electrical reactions in the muscles of the face were those of peripheral paralysis. Vasomotor phenomena, such as pallors, flushings, and the epistaxis and mucous hemorrhages referred to above, may be present in pontile lesions, as may also special temperature changes. These are probably due to implication of vasomotor and heat centres.

FIG. 436.



Microscopical appearances of a transection near the middle of the pons through the lesion of the case shown in Figs. 432, 433, 434, and 435.

CHAPTER X.

DISEASES OF THE GLOSSOPHARYNGEAL NERVE (MOTOR AND SENSORY DIVISIONS), AND OF THE PNEUMOGASTRIC, SPINAL ACCESSORY, AND HYPOGLOSSAL NERVES; SMALL GROSS LESIONS OF THE POST-OBLONGATA AND OBLONGATA-SPINAL TRANSITION; AND SOME FORMS OF BULBAR PARALYSIS.

ANATOMY AND PHYSIOLOGY OF THE GLOSSOPHARYNGEAL, PNEUMOGASTRIC, AND ACCESSORY NERVES.

Interrelations of the Glossopharyngeal, Pneumogastric, and Accessory Nerves.—The glossopharyngeus and vagus arise from a nucleus which histological research has not yet separated into the parts which belong to each of these nerves respectively. It is commonly taught, even in recent anatomical works, that the glossopharyngeus, the vagus, and the accessorius arise from a common nucleus, but the investigations of Grabower and Oppenheim seem to make it clear that the accessorius nucleus is distinct from the nucleus of the vagus. While a similar separation has not been determined for the glossopharyngeus and the vagus, it is at least demonstrable that the glossopharyngeal nucleus is in the more cephalic portion of the so-called vagoglossopharyngeal nucleus. One of the great root bundles in the postoblongata, usually spoken of as a vagoglossopharyngeal root, Obersteiner believes to be almost purely glossopharyngeal, holding that while some of the fibres of this root may belong to the vagus, the number of these is inconsiderable. The glossopharyngeus and the vagus supply sensory and motor fibres to adjacent and closely related regions, and the functions of these nerves in some cases interblend or have a certain degree of interdependence, as shown, for instance, in the relationship between such processes as those of deglutition and digestion or of deglutition and respiration. Many general works on neurology, and especially those in German, consider together affections of the vagus and accessorius under the head of vagusaccessorius diseases; some even go so far as to speak of glossopharyngeusvagusaccessorius diseases; but it will fulfil a better purpose to discuss their diseases separately, although the anatomy and physiology of these nerves may be treated of together.

Subdivisions of the Glossopharyngeal Nerve.—When discussing the affections of taste, the anatomy of the glossopharyngeus was to a large extent considered (pages 686–688), most attention being paid to its gustatory division. As there stated, it is generally conceded that the glossopharyngeus springs from three oblongatal

regions. According to Obersteiner, the three separate functions of the glossopharyngeal nerve, namely, taste, motility, and general sensation, may without hesitation be assigned to its three separate nuclei. The same author points out the anatomical similarity between the spinal descending root of the trigeminus and the great root of the glossopharyngeus, believing that the latter is also descending and, like the former, is concerned with impressions of common sensibility. It is sometimes spoken of as the slender column of Lockhart Clarke. It lies to the outer side of the glossopharyngeal and pneumogastric nerves and near the floor of the fourth ventricle, and is a slender column of longitudinal fibres which appear rounded on transection and very small in size. This column extends cephalad as far as the most cephalic portion of the nucleus of the glossopharyngeus, and caudad to the deep portion of the lateral column of the cord into the cervical region, although it is impossible to state the exact spinal termination of the bundle. Formerly it was called the ascending root of the vagus and glossopharyngeus nerves. By Meynert it was spoken of as the common ascending root of the lateral mixed system. In function it has been supposed to be in some way connected with the process of breathing; hence it has been termed the respiratory column of Krause. Section of this bundle on both sides arrests the respiratory movements. Spitzka has, however, denied the connection of this bundle with the vagus, and the views of Obersteiner have been previously given. About the level of the entrance of the glossopharyngeus into the postoblongata the fibres of this bundle may be observed in a transection to alter their course. In sections from slightly lower levels the fibres of the bundle are cut transversely, but in the region mentioned they bend horizontally, and may be distinctly seen passing towards the ventral border of the postoblongata. The origin, root fibres, and nuclei of the sensory portion of the glossopharyngeus have been sufficiently considered in the pages to which reference has just been made. Investigations making a clear distinction between the subdivision of the nerve which administers to taste and that which is related to common sensibility are not as yet forthcoming. The most that can be said with regard to the ganglia of the roots, the sensory root fibres, and nuclei, is that they are afferent, and are concerned both with taste and with common sensation, the motor portion, of course, being efferent. The peripheral course of the glossopharyngeus and its branches is given on pages 687 and 688.

Summary of the Functions of the Glossopharyngeus.—The glossopharyngeal nerve sends branches to the mucous membrane of the tongue, pharynx, and middle ear, as well as to the stylopharyngeus muscle, and possibly also to the middle constrictor of the pharynx. By its small superficial petrosal branch it furnishes secretory and vasodilator fibres (through the otic ganglion and the

auriculotemporal nerve) to the parotid gland. It is connected with the inferior maxillary division of the fifth (through the otic ganglion), with the facial, with the pneumogastric (its trunk and branches), and with the sympathetic. (Quain's Anatomy.) According to some observers, it is a motor nerve for the glossopalatinus, the levator veli palatini, and the azygos uvulæ, but for the last two at least the nerve supply is more probably from the vagus. It is generally conceded that the glossopharyngeus is the nerve of common sensibility for the upper part of the pharynx, and perhaps for the tympanic cavity and a part of the palatal region. Stimulation of the sensory fibres of the glossopharyngeus may excite movements of deglutition reflexly through the muscles of the palate and pharynx. Both the gustatory fibres and the fibres of common sensibility may through their stimulation excite reflexly a flow of saliva.

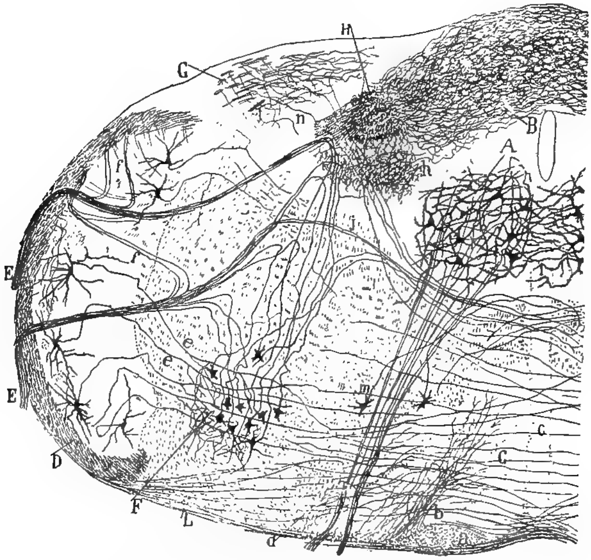
Ventral and Dorsal Subdivisions of the Nuclei of the Glossopharyngeal and Pneumogastric Nerves.—The glossopharyngeal and pneumogastric nuclei have in the first place *ventral* and *dorsal* subdivisions. The ventral cell nests give origin to the motor or efferent root fibres of these nerves, and taken together form the so-called *nucleus ambiguus*, or *common nucleus*. The dorsal nuclei are the end nuclei (the postoblongatal termini) of the sensory or afferent fibres of the vagoglossopharyngeal sensory nerve roots.

Dorsal Vagoglossopharyngeal Nucleus.—This nucleus is a long column of cells. At its most caudal portion it is dorsal to the hypoglossal nucleus at the point where the central spinal canal is still closed; more cephalad it is lateral to the nucleus of the hypoglossus, forming the *ala cinerea* of the floor of the fourth ventricle. This nucleus by some observers is differentiated into three parts for the glossopharyngeus, vagus, and accessorius respectively, but Turner believes that this segmentation is not certainly determined. It has long been taught that this dorsal nucleus is sensory, a view which has been confirmed by the more recent investigations of Ramón y Cajal, Turner, and others. Eisenlohr believes that this dorsal nucleus is the centre for the superior laryngeal nerve, and Holm that it is a respiratory centre. More exactly, Holm's conclusions are, (1) the centre for the trachiobronchial reflex is probably located in the dorsolateral division of the dorsal nucleus of the vagus,—the small ganglionic cells of the nucleus; and (2) the respiratory centre consists exclusively of the ventromesal division of the dorsal nucleus of the vagus,—the large celled group of the nucleus. Marinesco as a result of his general study of the subject concludes that the dorsal nucleus is not essential to respiration, but that it contains motor neurons for the muscles innervated by the pneumogastric, and is therefore a motor nucleus, although the cells are not of the same type as those of the nucleus ambiguus or of the hypoglossal nucleus. He calls the dorsal nucleus the nucleus of the

nonstriated muscles, in contrast to the nucleus ambiguus, which he designates as the nucleus of the striated muscles innervated by the pneumogastric, —the muscles of the larynx, for instance.

Nucleus Ambiguus.—According to Koelliker, this nucleus extends from the level of the lowest vagus fibres, proximally as far as the exit of the upper glossopharyngeal root fibres. It begins in about the same region as the hypoglossus nucleus, but extends farther cephalad than the latter. It occupies an anterolateral position in the postoblongata, and is usually roughly divided into three portions, the upper for the glossopharyngeus, the middle for the

FIG. 437.



Section through the bulb of a four-day old mouse, showing the nuclei and radicles of the glossopharyngeus, vagus, and hypoglossus: A, hypoglossal nucleus; B, commissural nucleus; C, olive; D, descending sensory root of the trigeminus; E, E, motor roots of the vagus and glossopharyngeus; F, nucleus ambiguus; G, terminal portion of the descending vestibular nucleus; H, transverse section through the fasciculus solitarius; L, fibres passing to the olive; a, pyramid; b, collaterals of the pyramid and of the white substance external to them; d, collaterals of the remains of lateral column; e, sensory collaterals to the nucleus ambiguus; f, recurrent fibres of the motor root which pass to the trigeminus root; j, crossed motor root fibres of the vagus and glossopharyngeus; h, collaterals of the sensory root of this nerve to the nucleus accompanying the fasciculus solitarius. (Ramón y Cajal.)

vagus, and the lower for the accessorius. It will presently be seen, however, that according to recent investigations the accessorius nucleus has no connection with the nucleus ambiguus. The nucleus ambiguus is composed of large motor cells from which pass long axis cylinders which take a bending course to reach the surface of the postoblongata, where they become the root bundles of the glossopharyngeus and the vagus. These cells and the course taken by their processes are beautifully shown in the illustration Fig. 437.

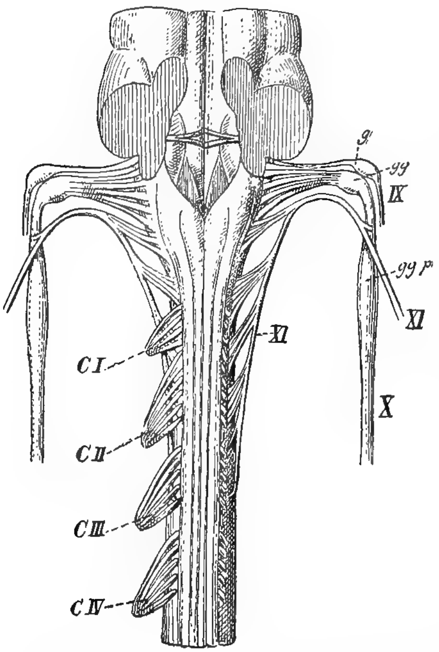
According to Turner, the nucleus ambiguus innervates among other structures the levator palati and internal thyroarytenoid muscles, and probably also the muscles of the pharynx. In 1879 Spitzka discovered deeply embedded in the oblongata in this region a cell nest to which he gave the name of *nidus laryngei*, near which is another nest of smaller elements, the *nidus pharyngei*. Springing from the nucleus ambiguus, the root fibres of the glossopharyngeal and pneumogastric nerves traverse the oblongata to emerge by the side of the prominence formed by the restis, in their course passing through the descending root of the fifth nerve. They have numerous filaments of origin, and it is difficult, if not impossible, to tell which extrabulbar roots belong to the ninth and which to the tenth nerve. It can be said with certainty only that the uppermost belong to the ninth and the lowermost to the tenth.

Central Neurons of the Motor Portion of the Glossopharyngeus and Vagus.—All the cerebral connections of the glossopharyngeus and vagus have not yet been traced. Their central neurons arise in some portion of the cortex, and probably pass near the pyramidal tract downward, reaching after decussation the nucleus ambiguus. Probably each portion of these nerves related to a special function has its own cerebral region of representation. The locations of the cerebral centres of laryngeal and pharyngeal movement have already been considered (see cortical localization, page 337). The vagal and glossopharyngeal neurons concerned with these movements doubtless arise in the portions of the cortex there indicated, namely, near the base of the precentral fissure for laryngeal movements, and just caudad of this location for the movements of the pharynx. Nothing of a positive character is known regarding cortical centres (if such centres exist) for movements of the œsophagus, and of the stomach, intestines, heart, and other viscera to which pneumogastric fibres are distributed.

Peripheral Course of the Vagus.—*General Course.* The peripheral course of the glossopharyngeus has been given in Chapter VII. (page 687). Superficially the pneumogastric nerve arises by numerous radicles from a groove between the olive and the restis, just below the so-called superficial origin of the glossopharyngeus (Fig. 438). The flat trunk formed by the union of its roots passes outward across the flocculus of the cerebellum, and leaves the cranium by way of the jugular foramen. It passes down the neck in the same sheath with the carotid artery. The right and the left vagus differ somewhat in the courses which their trunks take in the neck and thorax, a point that should be remembered in connection with the consideration of extrinsic focal lesions affecting the trunk of the nerve. The right vagus passes in front of the first portion of the subclavian artery, between the latter and the subclavian vein. It then runs downward on the side of the trachea to reach the posterior

aspect of the right bronchus. The left vagus enters the thorax behind the left common carotid artery, between the latter and the left subclavian artery. Near the arch of the aorta it bends suddenly backward and crosses the root of the subclavian artery on the left side of that vessel under cover of the pleura. Crossing the left side (in front) of the transverse part of the arch of the aorta, it bends backward to reach the posterior aspect of the left bronchus. (Morris's Anatomy.) The right vagus is distributed on the posterior surface of the stomach, and the left vagus on its anterior surface.

Fig. 438.



Apparent origins of the spinal accessory, pneumogastric, and glossopharyngeal nerves: *g*, jugular ganglion of the glossopharyngeus; *gg*, ganglion of the root of the vagus (ganglion superius, ganglion jugulare); *gg p*, ganglion of the trunk of the vagus (ganglion inferius, plexus nodosus, plexiform ganglion). The Roman numerals indicate the cranial nerves and the cervical nerves in their order. (After Van Gehuchten.)

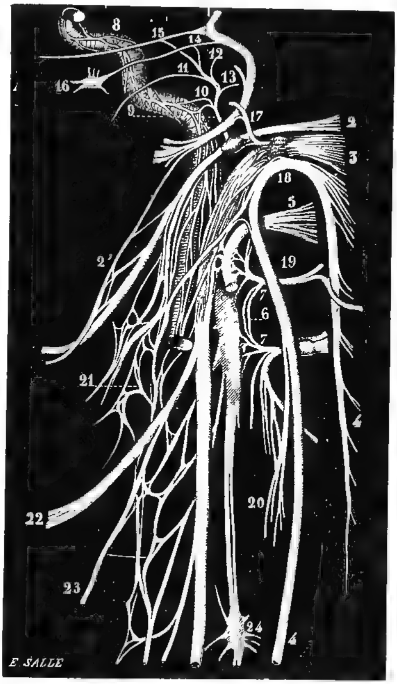
Ganglia of the Vagus. The vagus has on its trunk two ganglia, an upper, or *ganglion of the root* (*ganglion superius, ganglion jugulare*), and a lower, or *ganglion of the trunk* (*ganglion inferius, plexus gangliformis, plexus nodosus*). The former is placed within the jugular foramen opposite the jugular ganglion of the glossopharyngeus, and the ganglion of the trunk is situated a little lower on the trunk of the nerve (Fig. 438). The superior cervical ganglion of the sympathetic is behind and a little external to the vagus, while the glossopharyngeus is in front of it. A portion of the spinal accessory nerve joins the ganglion of the trunk, some of the fibres running over the ganglion into the pharyngeal and superior laryngeal branches of the vagus.

Communicating Branches of the Vagus. The vagus has important connections with the glossopharyngeal, the spinal accessory, the hypoglossal, the cervical sympathetic, and the first two cervical nerves. (Fig. 439.) A twig from the pneumogastric joins the glossopharyngeal nerve immediately below the petrous ganglion. Fine filaments pass from the accessorius to the ganglion of the root of the vagus within the jugular foramen, and below, the remainder of the accessorius joins the ganglion of the root. Fine filaments also pass

between the ganglion of the trunk of the vagus and the hypoglossal nerve as the latter winds around this ganglion. A few twigs pass between the superior cervical ganglion of the sympathetic and the ganglion of the trunk. The branches of distribution of the vagus also communicate extensively with the sympathetic system at lower levels. An inconstant twig passes from the loop formed by the anterior primary divisions of the upper two spinal nerves to the ganglion of the trunk of the vagus.

Branches of Distribution of the Vagus. For a detailed description of the method of distribution of the numerous branches of the vagus, works on anatomy should be consulted. Reference will here be made only to a few facts regarding the branches most important to the clinician. The branches of distribution as usually given are the meningeal, auricular, pharyngeal, superior laryngeal, inferior laryngeal, cardiac, pulmonary, esophageal, and abdominal. The meningeal branch probably contains chiefly sensory and vasomotor fibres. The auricular branch is in part a sensory nerve, by its subdivisions supplying the posterior part of the auditory meatus and the adjoining part of the external ear. Another branch runs with the posterior auricular branch of the facial. The pharyngeal branch is one of the principal motor nerves of the pharynx. After subdivision into numerous filaments, it communicates with other filaments from the glossopharyngeus, the external branch of the superior laryngeal, and the sympathetic, to form the pharyngeal plexus, from which branches are distributed to the mus-

FIG. 439.



The last four cerebral nerves, the facial nerve, the sympathetic, and the upper two cervical nerves. 1, facial nerve; 2, glossopharyngeal; 2', anastomosis between a branch of the facial and the glossopharyngeal; 3, vagus; 4, accessory; 5, hypoglossal; 6, first cervical ganglion of the sympathetic; 7, first and second cervical nerves; 8, cavernous plexus of the sympathetic on the internal carotid artery; 9, tympanic nerve from the petrous ganglion of the glossopharyngeal; 10, its connection with the carotid plexus; 11, branch to the Eustachian tube; 12, 13, branches to the oval and round windows of the ear; 14, 15, branches joining the small and superficial petrosal nerves; 16, otic ganglion; 17, auricular branch from the jugular ganglion, connected by filaments with the petrous ganglion and the facial nerve; 18, anastomosis of the accessory with the vagus; 19, anastomosis of the first cervical nerve with the hypoglossal; 20, anastomosis of the second cervical nerve with a branch of the accessory; 21, pharyngeal plexus; 22, superior laryngeal nerve; 23, its external branch; 24, second cervical ganglion of the sympathetic. (After Hirschfeld-Sappey.)

cles and mucous membrane of the pharynx. The superior laryngeal is the chief nerve of sensation to the larynx, although it also supplies branches to the cricothyroid and arytenoid muscles. The inferior or recurrent laryngeal nerve is the great motor nerve of the larynx. The cardiac, pulmonary, esophageal, and abdominal branches of the vagus contain both sensory and motor fibres. Edinger speaks of the vast number of sensory fibres which are found in all parts of the body,—in the liver, kidneys, lungs, etc.,—fibres which are necessary for the reflexes on which life itself depends. The vagus nucleus in the fish forms a lobe, probably because the nerve to which it gives origin is distributed also to the external surface of the body like any sensory spinal nerve.

Nerve Supply to the Heart.—The nerves to the heart come from the cardiac plexuses, and are derived both from the vagus and from the gangliated system. Among the most important of these nerves is the *depressor nerve of Cyon.* So-called *accelerator nerves*, which are said to have their origin in the cervical spinal cord, reach the heart by the last cervical and the first thoracic ganglion of the sympathetic system. The depressor nerve in the rabbit arises by one branch from the superior laryngeal, and usually also by a second root from the trunk of the vagus itself, and passes down the neck to the cardiac plexus. In the horse and in man fibres analogous to those of the depressor re-enter the trunk of the vagus to descend to the heart. This depressor nerve is an afferent (sensory) nerve. The ordinary rhythmical movements of the heart are associated with the activity of nerve ganglia which exist in the substance of the heart, the so-called intracardiac ganglia. The true inhibitory nerve of the heart arises from the inferior cardiac nerve, and is distinct from the depressor nerve of Cyon. Its stimulation slows the action of the heart and augments the force of its beat.

Nucleus of the Accessorius.—As stated, the vagus and accessorius are usually described as originating in a continuous nucleus, the lower portion of the nucleus ambiguus. Obersteiner says that the roots of the accessorius vagi (the proximal roots of the accessorius) have exactly the same origin as the vagus itself, from which they cannot be separated within the brain. The observations of Grabower and Oppenheim have, however, thrown great doubt upon the origin of the vagus and the accessorius in one nucleus. Oppenheim thinks that Grabower has established the fact that the motor innervation of the larynx is entirely from the vagus. The conclusions of Grabower in his last paper (1896) were based upon the lesions found in a case of tabes, which were degeneration of the left recurrent laryngeal nerve, of the extrabulbar portion of the roots of both tenth nerves, of the roots of both ninth nerves, and of the spinal roots of both fifth nerves. The nuclei of the third, fourth, and sixth nerves were also atrophied. The roots of both eleventh

nerves and the nuclei and intrabulbar portion of the roots of the tenth and eleventh nerves were perfectly preserved. The symptoms were disturbances in the territory of the trigeminus, oculomotorius, abducens, glossopharyngeus, acusticus, and the laryngeal branches of the vagus, but there were no symptoms referable to the accessorius distribution. The laryngoscope showed a complete paralysis of the left recurrent laryngeal nerve. Grabower has shown that an encephalic accessorius nucleus does not exist, that from where the nucleus of the accessorius terminates to where the motor nucleus of the vagus (nucleus ambiguus) begins is a space of more than nine millimetres, and that there is not the slightest connection between the vagus and accessorius nuclei.

Peripheral Course of the Spinal Accessory Nerve.—The so-called bulbar portion of the spinal accessory, which from recent investigations, as stated, should be considered a part of the vagus, consists chiefly of very fine nerve fibres, while the spinal portion is composed almost entirely of large fibres. In the postoblongata the former portion has the same origin as the vagus, whereas the distal roots arise from cells which have no connection with the vagal nucleus. The nucleus of the spinal portion of the spinal accessory nerve is constituted of a continuous series of cells which forms a part of the ventrolateral group of cells of the ventral horns of the cord. The fibres arising from this group of cells pass almost directly outward and backward, others for a certain distance longitudinally, and then outward and backward to emerge at the lateral columns of the cord, usually between the fifth cervical nerve and the first. In exceptional cases the lowest nerve filaments which make up the spinal portion of the accessory nerve may pass out from the cord as low down as the seventh cervical nerve. The different filaments composing the nerve are not exactly in the same anteroposterior line, the higher of these filaments arising closer to the dorsal nerve roots, with one or two of which they may be connected. These widely separated roots come together to form a rounded bundle, which, taking a cephalic course, and augmenting in bulk as it passes cephalad, traverses the foramen magnum and reaches the cranial cavity. Just after entering the skull it turns outward to pass into the middle compartment of the jugular foramen, where it is combined in the same sheath of dura as the vagus, and also with the so-called bulbar portion of the spinal accessory. These three nerve bundles form a single trunk in this portion of the jugular foramen. It now turns downward, outward, and backward to pass out of the skull again between the occipital artery and the internal jugular vein. The spinal portion of the nerve passes under covering of the sternocleidomastoid muscle, to which it gives deep branches, communicating here with fibres direct from the second cervical nerve. Emerging at the posterior border of the sternocleidomastoid muscle, the nerve crosses

obliquely the posterior triangle of the neck to the under surface of the trapezius, where it is joined by branches from the third and fourth cervical nerves, forming with them the so-called subtrapezoidal plexus. Both the trapezius and the sternocleidomastoid muscle, it will be seen, are supplied from the spinal portion of the spinal accessory and to a much less degree directly from the cervical nerves.

AFFECTIONS OF COMMON SENSIBILITY DUE TO DISEASE OF THE GLOSSOPHARYNGEUS.

The relations of the glossopharyngeus to disorders of taste having been fully considered, it will only be necessary here briefly to discuss affections of common sensibility and of motility due to disease of this nerve. As has been stated (page 937), it is generally conceded that the glossopharyngeus is the nerve of common sensibility for the upper part of the pharynx and perhaps for the tympanic cavity and a part of the soft palate. If this be true, disease in any portion of the pathway for common sensibility of this nerve should cause anesthesia of these regions; but accurate clinical data are wanting. It is probable that in either the jugular or the petrous ganglion of the glossopharyngeus are situated the cells of origin of the glossopharyngeal nerves of common sensibility, and if Obersteiner's view is correct it is also probable that in the general nucleus for the glossopharyngeus a separate cell nest is the end nucleus for the peripheral neurons. Destructive disease of the ganglion of origin or of the end nucleus would, of course, cause anesthesia of the parts to which the nerve is supplied, as would also disease of any portion of the peripheral nerve trunk; but cases have not been recorded in which restricted anesthesia has indicated lesion of such a limited portion of the glossopharyngeus. Irritation of the ganglia, root fibres, or peripheral distribution of the nerve would cause hyperesthesia or neuralgia of the upper portion of the pharynx, the tympanic cavity, and a part of the palatal region. Another symptom would be absence of reflex irritability of the pharyngeal mucous membrane. The only well studied diseases in which pharyngeal anesthesia is a marked symptom are the postinfectious paralyses, and especially postdiphtheritic paralysis; and even in these affections the anesthesia is not usually an isolated symptom, and is commonly not restricted to what is supposed to be the glossopharyngeal sensory distribution.

PARALYSIS IN THE DISTRIBUTION OF THE GLOSSOPHARYNGEUS.

While great uncertainty still exists as to the exact distribution of the motor portion of the glossopharyngeus, assuming that it is the motor nerve for the stylopharyngeus muscle and possibly also for the

middle constrictor of the pharynx, it follows that lesion of the motor nucleus in the postoblongata, or of the peripheral distribution of the nerve, will cause paralysis of these muscles. Interference with deglutition will be the most prominent symptom of this paralysis. Usually the other muscles, like the levator palati, azygos uvulæ, and glossopalatinus, are paralyzed with the middle constrictor of the pharynx and the stylopharyngeus, so that from any actual records of cases it is impossible to separate the symptoms which are solely attributable to the glossopharyngeal paralysis. As the pharyngeal muscles, besides the part played by them in swallowing, also exert an important influence in vocal modulation, and especially as regards the higher tones, changes in the voice will be other symptoms. In all the recorded cases of disease of the sensory and motor portions of the glossopharyngeus (not including the gustatory division) motion and common sensibility have been affected together. This, as is well known, is particularly the case in the diphtheritic paralyses. It is a somewhat striking fact that in the postinfectious diseases taste is usually not affected, which argues in favor of separate nuclei for the parts of the nerve related to taste and common sensibility. In chronic bulbar paralysis the motor glossopharyngeal nucleus is frequently involved. Lloyd suggests that one reason why so little has been observed of symptoms of glossopharyngeal paralysis is that all symptoms produced by lesions of this nerve are deepseated, and therefore readily overlooked unless searched for with the most careful tests. "A patient, for instance, might readily have an anesthesia of one side of the pharynx and loss of taste on one side of the posterior third of the tongue, without this being very perceptible to himself or very readily detected by his physicians. It is doubtful whether the unilateral lesion would seriously interfere with the act of swallowing. This act, being largely reflex, no doubt depends to some extent upon the integrity of the sensory fibres in the glossopharyngeal nerve, but the irritability might readily be blunted, we may suppose, or even abolished on one side, without the act of swallowing being more than slightly embarrassed. It is impossible to say, owing to lack of observation, to what extent speech, especially the formation of gutturals, might be impaired by complete paralysis of the glossopharyngeal nerve. It is probable that this nerve conveys the muscular sense from parts of the pharynx and fauces, and the abolition of this we might suppose would cause some incoordination of the movements necessary for speech; but of this we have no positive clinical evidence. As the nerve supplies common sensation to and about the epiglottis, its paralysis would tend to cause choking by permitting small particles of food to find their way into the windpipe." Nothing need be said about the treatment of affections of the glossopharyngeal nerve. The principles of treatment are the same as those for the other cranial nerves.

DISEASES OF THE VAGUS.

Diseases of the Vagus as a Whole.—It has been shown that the pneumogastric is a great sensory and motor nerve, and that some of its branches (sensory or motor) act as special nerves to the great viscera of the thorax and abdomen,—to the heart, lungs, stomach, intestines, liver, and perhaps other organs. The separate sensory and motor affections of this great nerve will be presently considered, but it is necessary here to say a few words about lesions affecting either the nerve or its nucleus in its entirety. It is, of course, evident that a lesion absolutely destructive of either the nucleus or the trunk of a nerve which is the source of innervation of organs so important to life as the heart and the lungs would cause death, but in not a few instances lesions are of such a character and of such an extent as to involve in greater or less degree the entire nucleus or entire nerve trunk without such fatal result. Such a lesion will cause a syndrome composed of sensory, motor, and visceral symptoms irregularly grouped. The subsequent presentation of the sensory, motor, and special symptoms of lesions of the vagus will be sufficient to indicate what might be expected in the case of a lesion irregularly involving the entirety of the nucleus or trunk of the nerve, but reference may be briefly made to a few of these cases. Diseases involving the pneumogastric as a whole are gross focal lesions, chronic degenerative diseases, or neuritis. Many cases of focal lesion involving the vagus have been put on record. Mays collected from literature a very large number of these cases, which included aneurisms, tumors, enlarged glands, wounds accidental and surgical, degeneration, and inflammation. The symptoms recorded in these and in cases collected by others are those both of irritation and of paralysis, in various combinations,—such phenomena as pharyngeal and laryngeal anesthesia and hyperesthesia, neuralgias of the same regions, angina pectoris, faucial paralysis and spasm, pharyngeal and laryngeal paralysis and spasm, cardiac neuroses, such as tachycardia, brachycardia, cardiac arhythmia, respiratory neuroses, and nervous disorders of the digestive apparatus. Cheyne-Stokes breathing is probably due to interference with the function of the pneumogastric centres. The epigastric aura of many epileptic fits, and the globus hystericus, are probably also to be referred to disturbance of the vagus. (Gowers.) Numerous instructive instances of the results of lesions interfering with the pneumogastric in its entirety have been recorded, as where the nerve has been tied by surgeons with the left carotid, when respiration and cardiac action are soon arrested. True paralysis of the heart and of respiration sometimes rapidly supervenes as the result of powerful impression made by toxic or infectious agents upon pneumogastric centres. In the comparatively numerous recorded cases of progressive degeneration of the vagal

nucleus in the course of diseases like tabes, one function of the nerve after another is lost, such symptoms as laryngeal and pharyngeal anesthesia and paralysis usually being prominent long before a fatal issue is reached.

Vagal Neuritis.—The vagus is at times attacked by neuritis. The presence of multiple neuritis was noted in four out of ninety-eight cases of disease with degeneration of the pneumogastric nerve tabulated by Mays to show the relation between disease of the vagi and consumption and other pulmonary affections. In the nervous wards of the Philadelphia Hospital, where alcoholic multiple neuritis is frequently seen, the patients occasionally die with respiratory and cardiac symptoms which point to inflammatory or toxic involvement of the vagi or their centres. Several such cases have fallen under my observation, and Lloyd refers to similar cases. He speaks, for instance, of a patient suffering with alcoholic multiple neuritis in all four extremities, with wandering delirium and tachycardia, who died suddenly from heart failure. He refers to the fact that in alcoholic multiple neuritis tachycardia is a very common symptom, the heart often beating at the rate of from 100 to 120 or even more. In these cases the heart is rebellious to cardiac stimulants, such as digitalis and strophanthus. Sharkey also has called attention to the fact that the vagus is disturbed in multiple neuritis, and this disturbance gives rise to marked and even fatal cardiac symptoms. Allyn records a case of extensive multiple neuritis with involvement of the vagus in which the patient recovered. Among the pneumogastric symptoms were pain and oppression over the heart, and collapse, with cool moist skin and extremely weak and irregular pulse. According to Sharkey, a few observations have shown that the terminal distributions of the nerves of the heart are liable to alterations which probably play an important part in some cases of serious cardiac disease. He suggests that the sympathetic as well as the vagus may be involved, and that such involvement may account for the visceral neuralgias and for disturbances of secretion and metabolism. Ferguson has reported the case of a woman aged forty who a short time after an attack of influenza had passed off began to have paroxysms of pain of the most agonizing character. She died after a period of great suffering which lasted ten weeks, and the postmortem examination of the nerves and the ganglia throughout the abdomen showed them to be highly inflamed. Marked degeneration of the nerve tissue was found on microscopical examination. In a case of influenza recorded by Sansom the patient had shooting pains in the epigastrium. The case, although alarming, ended in recovery. Vagal neuritis was probably present. The phrenic as well as the vagus, and even more frequently than the latter, is affected. Close watch should always be kept on the action of the heart and the character of the breathing in multiple neuritis. (Allyn.)

DISEASE OF THE SENSORY PORTION OF THE VAGUS.

Varieties of Pneumogastric Sensory Disease.—The sensory branches of the pneumogastric, wherever situated, may be affected by disease of the same character as affects other sensory nerves. As in the case of the sensory division of the trigeminus, for instance, we may have hyperesthesias, anesthesias, and paresthesias, symptomatic or essential neuralgias, and special forms of sensory disturbance.

Pneumogastric Neuralgias.—Every portion of the sensory supply of the pneumogastric has been in recorded cases the seat of neuralgia,—pain which has not been traceable to an organic cause, or at least not to a gross lesion directly irritating the nerve or its centres. In rare cases such pain affects the posterior portion of the auditory meatus and adjacent portions of the ear. Neuralgia of the pharynx and soft palate is an affection of somewhat rare occurrence, and is probably due to a disturbance of both the sensory portion of the glosso-pharyngeal nerve and the vagus. The various regions supplied by the sensory branches of the superior laryngeal nerve may also be the seat of severe neuralgias. The "laryngeal crises" are not, like most of the other so-called "crises" of tabes, wholly of a sensory, or at least of a painful, character. The effects of these crises are chiefly spasmodic and respiratory, but severe laryngeal pain might be a part of their symptomatology. In cases of tabes which have been examined at or near the time of such laryngeal crises the mucous membrane of the larynx immediately before the attacks has been found extremely hyperesthetic. The patient sometimes feels a scalding sensation or experiences the sensation of a foreign body lodged in the larynx. (Ross.) Severe paroxysmal gastralgia (stomach pain) may be a pure pneumogastric neuralgia, but such forms of neuralgia are extremely rare, except as they occur in the gastric crises of tabes. While pain in the stomach may be directly traceable to irritation of terminal filaments of the pneumogastric, it is, as a rule, caused by lesions of the mucous membrane or other nonnervous structures of the stomach. The gastric crises of tabes are among the most distressing phenomena of this disease; not infrequently they are early manifestations. The torturing pains of the gastric crises of tabes are probably due in large part or altogether to sclerotic degeneration of the dorsal nucleus of the vagus. They have been occasionally attributed to sympathetic disease. The intercostal and spinal sensory nerves associated with the pneumogastric may take part in the production of the pain. In the case recorded by Grabower the patient had violent crises both laryngeal and gastric, the latter becoming so excessive that the patient vomited almost all that he ate and died from exhaustion. In this case it will be remembered (see page 942) that among other

lesions was found degeneration of the left recurrent laryngeal nerve and of the extrabulbar portion of the roots of both tenth nerves.

Angina Pectoris of Neural Origin.—The large majority of cases of angina pectoris are due to various organic affections of the heart, the pericardium, or the aorta, or to sclerosis of the coronary artery, aneurism of the arch of the aorta, chronic aortic stenosis or insufficiency, fatty degeneration of the heart muscle, or chronic adhesive pericarditis. Even in these cases the disease may in a sense be regarded as a pneumogastric neurosis, as the pain, usually excruciating in character, is due to irritation of the cardiac sensory fibres. It will be remembered that the depressor nerve of Cyon is a sensory nerve. In rare cases the symptoms of angina pectoris result from purely neural lesions, such as a neuritis of the vagus, irritative diseases of the vagal centre, or a toxic agent affecting these centres. Leroux has recorded a case in which angina pectoris was present, and in which the autopsy showed the right vagus compressed by enlarged glands. Cases have been recorded in which anginal attacks were associated with small tumors of the vagus. When a tumor involving the cardiac plexus causes angina it is just as correct to assign the lesion to the vagus as to the sympathetic, as the cardiac plexuses are formed by the union of nerve branches from both sources. Besides the pain which is the dominant feature of angina, other symptoms, such as changes in the rapidity and rhythm of the action of the heart, with a sense of distress, suffocation, or dissolution, are referable to disturbances of the pneumogastric.

Laryngeal Hyperesthesia.—Hyperesthesia of the mucous membrane of the larynx, which must, of course, depend upon direct or reflected irritation in the sensory laryngeal branches of the vagus, is most commonly the result of inflammation of the laryngeal structures. It is frequently present in carcinomatous, tubercular, and various forms of chronic catarrhal disease of the larynx. In cases of chronic alcoholism it is of comparatively frequent occurrence, and in these cases probably depends, in some instances at least, upon a neuritis of alcoholic origin, pain as well as hyperesthesia sometimes being present. When the sensibility of the larynx is heightened, various reflex phenomena, such as coughing, or local spasm, or even epilepsy, may occur.

Laryngeal Anesthesia.—Laryngeal anesthesia occurs as an independent affection due to a focal lesion in the course of the sensory branches of the superior laryngeal nerve; it may be due to agents acting upon the nerve centres or nerve termini; or it may be one of the symptoms of tabes, here being due to degeneration of the laryngeal portion of the vagal nucleus. Partial anesthesia is not infrequently simply a part of the symptomatology of a chronic inflammation of the larynx. Anesthesia following the infectious diseases, as diphtheria, is most frequently dependent upon the action

of a toxine upon the nerve centres, tracts, or termini. Anesthesia of the larynx is not infrequently one of the stigmata in hysteria. The extraordinary tolerance with which hysterical women bear examinations of the pharynx and larynx is well known both to laryngologists and to neurologists. The presence of laryngeal anesthesia is determined by such examinations, and also by certain symptoms which are the results of the existence of this anesthesia. Reflex coughing, for instance, does not take place, and catarrhal secretions and foreign matter of any sort are more likely to be retained in the larynx and thus give rise to inflammation, with all its symptoms and sequences.

Gastric Anesthesia.—Anesthesia in the territory of the gastric branches of the vagus gives rise to polyphagia, a condition in which an unusual quantity of food must be taken before the feeling of hunger is appeased, or in which the feeling of repletion is never obtained however much food is taken. Experiments have shown that on section of the vagi animals continue to eat until the esophagus is filled with food. (Ross.)

Pneumogastric Paresthesia.—Almost any form of perverted sensation may be present in the sensory domain of the pneumogastric. Uncomfortable and strange sensations in the region of the stomach, liver, or spleen, sensations of tingling, of burning, or of irritation of any sort in the larynx or trachea, a constant tendency to swallow, or a feeling as if swallowing were continually taking place, the sensation of a foreign body in the larynx,—these and many other paresthesias may be due to local organic lesions, but in some instances they are due to functional or organic disorders of the sensory division of the vagus.

Treatment of Vagal Neuritis and Sensory Diseases of the Vagus.—If the existence of neuritis of the pneumogastric is recognized, great care should be taken not to push too far those remedies, like the salicylic compounds and coal tar products, which are so efficient in relieving the symptoms of ordinary neuritis. The probability of paralysis of cardiac action and respiration must be taken into account, and remedies like digitalis, strophanthus, ammonium carbonate, and even alcoholic stimulants may need to be used. Cardiac tonics and stimulants do not seem to be as efficient under these circumstances as they are ordinarily. With this caution, the treatment of vagal neuritis will be that of neuritis elsewhere,—the use of mercury, the iodides, the salicylates, strychnine, and narcotics and sedatives sufficient to give the patient reasonable comfort. Anesthesia in the distribution of the pneumogastric is to be treated by electrical applications when the anesthetic parts can be reached in this way, as in cases of pharyngeal and laryngeal anesthesia, and in other cases by constitutional remedies or measures designed to relieve the causative focal lesions. For the electrical treatment of the pharynx and larynx special electrodes may be required.

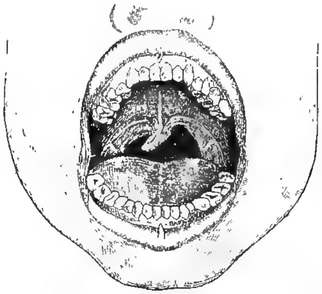
DISORDERS OF THE PALATAL AND PHARYNGEAL MUSCLES SUPPLIED BY THE VAGUS.

The pneumogastric supplies motor fibres to the voluntary muscles of the soft palate, with perhaps one or two exceptions. Views differ as to the supply to the tensor palati, and possibly some other muscles of the pharynx. According to comparatively recent investigations, the motor fibres of the levator palati are contained in the roots of the vagus. Good authority, however, as already shown, favors the view that the tensor palati is supplied by the fifth. The facial nerve in all probability does not take any part in the motor innervation of the palate. It is very commonly stated that of the muscles of the palate the levator palati and azygos uvulæ, at least, are supplied by the facial nerve, and some clinical records apparently support this contention. Cases of peripheral facial paralysis in which the levator palati and azygos uvulæ were paralyzed more or less completely have been carefully recorded by competent observers. An illustration of the combined paralysis of the levator palati and azygos uvulæ is given in Fig. 440. Complete peripheral facial paralysis was present in this case, which is reported by Sanders. The patient was also deaf in the ear of the paralyzed side. The symptoms therefore pointed to a peripheral lesion, but probably one which was intracranial or at least proximal to the position of the geniculate ganglion. If, as I believe, the nerve-supply of the velum palati and azygos uvulæ is from the vagus, the best explanation of a case like that of Sanders is that the branch from the vagus joins the facial and travels with it to the position of the geniculate ganglion, then passing by way of the great superficial petrosal nerve to its final destination. A gross lesion, therefore, involving the facial trunk anywhere from its superficial origin to the geniculate ganglion would also implicate the vagal branches to the velum palati and azygos uvulæ, and probably also the auditory nerve, giving just such a combination of symptoms as is recorded in the case reported by Sanders. When a lesion producing facial paralysis is distal to the geniculate ganglion, it will probably be found that the paralysis of the velum palati and azygos uvulæ does not form a part of the syndrome. Besides the muscles of the soft palate, the vagus also supplies the pharyngeal muscles, except those already considered, which derive their supply from the glossopharyngeus. It is also the chief motor nerve of the larynx. The cricothyroid muscles, which may be regarded as accessory to the thyroarytenoids, are supplied by the superior laryngeal nerve, and the other laryngeal muscles are supplied by the inferior or recurrent laryngeal. Commonly paralyses of the voluntary muscles of the mouth and throat supplied by both the glossopharyngeus and the vagus occur conjointly. This is, of course, what would be expected when it is remembered that these nerves have a common nucleus,

or at least nuclei in such close contiguity that they have not yet been anatomically separated, that their roots are blended at their superficial origin, and that the first portions of their peripheral course are largely the same. Degenerative nuclear disease will, therefore, most likely involve both the glossopharyngeal and the vagal nuclei if we regard these as distinct; and focal lesions either of the substance of the postoblongata or of its surface or its membranous or bony envelopes will to a greater or less extent invade both nerves. It is for this reason that the symptomatology of pharyngeal paralysis (*dysphagia paralytica*) is usually that of both vagal and glossopharyngeal disease. This form of paralysis has already been partly described under affections of the motor portion of the glossopharyngeus, where it has been shown that when not due to focal lesions it is most frequently a sequence of an infectious disease, like diphtheria, or a part of a degenerative affection, like tabes or bulbar paralysis. In this connection, therefore, it will only be necessary to say that paralytic dysphagia is a pneumogastric disease so far as the muscles concerned are distinctly supplied by the pneumogastric,—in other words, so far as they are supplied by the branches of the pharyngeal plexus in general which belong to the pneumogastric. Paralysis of the faucial muscles may be caused by hypertrophic tonsils. Paresis of the velum palati may be caused by enlarged cervical glands and by hypertrophic tonsils, affections which, according to Rethi, are more frequent than is commonly supposed. It is important to remember that degeneration of the azygos uvulæ on one side may be present, causing deviation of the uvula, without nerve lesion. If the faucial paralysis is limited to the pneumogastric supply (that is, does not implicate the glossopharyngeal muscles), the symptoms will be in part those of a general pharyngeal paralysis. If bilateral, owing to the involvement of the palatal muscles, the voice will become more or less nasal, the patient will have a sense of fulness in the mouth, the uvula will be inert and probably will hang down so as to touch the surface of the posterior part of the tongue. Some difficulty of deglutition will be present, but it will not be of the same character as that which is present in general pharyngeal paralysis. A few words need to be said about the features assumed by the paralysis when special palatal muscles or groups of muscles are involved. When the levator palati is paralyzed alone, the soft palate on the affected side is pendent and inert, and irritation does not cause the retraction and upward arching of the uvula, which, however, becomes tense laterally through the action of the tensor palati, its posterior edge being pulled somewhat downward by the action of the palatopharyngeus. The effect of combined paralysis of the levator palati and azygos uvulæ is shown in the illustration (Fig. 440) to which reference has been made above. The half of the velum on the paralyzed side is lower, but is not laterally displaced. With

regard to the position of the uvula, reports differ. According to Sanders, as illustrated in Fig. 440, the tip of the uvula is turned towards the paralyzed side; and this writer reports several other well studied cases in which the uvula was affected in a similar manner. Other cases, however, have been recorded in which the deviation was towards the nonparalyzed side, and it is difficult to account for these discrepancies. Probably many of the observations have been carelessly made. The explanation of the distortion towards the paralyzed side offered by Sanders is that the palatopharyngeus muscle, being unopposed by the levator palati, exerts a greater effect in drawing the uvula towards the side of the paralysis. When the levator and tensor palati muscles are both paralyzed, the velum is more pendent than when the paralysis affects the former alone, and the velum is also laterally displaced by the action of the tensor of the opposite side. Irritation of the soft palate on the affected side causes no retraction. Speech is decidedly nasal, and fluids regurgitate through the nose. Paralysis of the palotopharyngeus causes an altered appearance of the isthmus of the fauces. The posterior pillars are widely separated and immobile. Spasmodic affections of the muscles of the palate and pharynx supplied by the vagus are rare. It is possible, however, to have such spasm from a tumor or other irritative lesion affecting the nuclei or the branches of the vagus which go to the muscles under consideration. Pharyngeal spasm is also sometimes an accompaniment of acute or subacute pharyngitis. Clonic spasm or at least twitching movements of the soft palate have been observed in advanced cases of paralysis agitans. (Ross.) Pharyngeal and palatal spasm is sometimes a functional or an hysterical phenomenon. Occasionally it takes the form of a pharyngophobia. The patient has an obsession as regards swallowing, with perhaps some spasmodic phenomena in the pharyngeal muscles. Spasm of the pharyngeal muscles supplied by the vagus and glossopharyngeus is a part of the symptom picture of hydrophobia and pseudohydrophobia. Courmont has observed a case of veritable tonic spasm of the pharynx absolutely analogous to laryngeal crises, but without laryngeal, esophageal, or gastric phenomena. The symptoms were so intense that alimentation became impossible and death appeared imminent. The manifestations ceased abruptly under the influence of suspension, and hence it was inferred that the spasms must have been due to peripheral lesion.

FIG. 440.

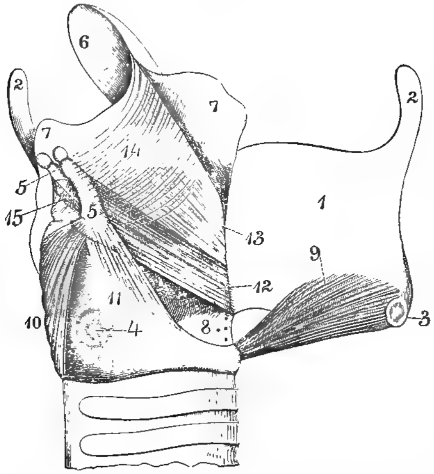


Distortion of the uvula in a case of peripheral paralysis of the right side of the face. (After Sanders.)

LARYNGEAL PARALYSES.

Varieties.—The larynx has a somewhat complicated system of muscles and cartilages (Fig. 441), with a comparatively simple neural supply, which, as already shown, is by two branches of the vagus, namely, the superior laryngeal and the inferior or recurrent laryngeal nerve. Laryngeal paralysis results from destructive lesions or functional disturbances of these nerves, their nuclei, or their corticobulbar pathway. It may be a *superior laryngeal paralysis* or an *inferior laryngeal paralysis*, and these neural types may have several subdivisions according to the degree of involvement of the nuclei or branches of these nerves. The paralysis may be *complete*, or it may be *incomplete*, *acute* or *chronic*, *unilateral* or *bilateral*. Another subdivision of laryngeal paralyses is into *respiratory* and *phonatory* types. A comparatively limited number of laryngeal movements is required in the act of breathing. In order that air may be inspired, it is simply necessary that the glottis should open. The posterior cricoarytenoid muscles are "glottis openers," and cricoarytenoid paralysis is therefore sometimes spoken of as pure laryngeal respiratory paralysis. After inspiration the cricoarytenoids relax, allowing the glottis to open and the air to pass out. Expiration is a passive and inspiration an active phenomenon. Respiration is largely reflex and involuntary; but phonation is a true voluntary act and chiefly connected with expiration. In phonation the vocal cords are apposed near the median line, the muscles taking part in this action being the lateral cricoarytenoid and the interarytenoid (arytenoid) muscles, supplied by the inferior or recurrent laryngeal nerve. In this act it is necessary that the vocal cords should be made tense. This tension is regulated by the actions of the posterior cricoarytenoid muscle, supplied by the superior laryngeal, and the thyroarytenoid muscle, supplied

FIG. 441.



Muscles of the larynx: 1, right ala of the thyroid cartilage, turned forward; 2, superior horns; 3, inferior horn, marked by the cricothyroid articulation; 4, the other portion of the latter on the side of the cricoid cartilage; 5, arytenoid cartilage, surmounted by the corniculate cartilage; 6, epiglottis; 7, the two laminæ of the aryepiglottic fold, separated so as to expose the muscles; 8, the lower part of the vocal membrane; 9, cricothyroid muscle; 10, posterior cricoarytenoid muscle; 11, lateral cricoarytenoid; 12, thyroarytenoid; 13, thyroepiglottic muscular fibres; 14, aryepiglottic muscular fibres; 15, arytenoid muscle. (Leidy.)

by the recurrent laryngeal. A phonatory paralysis is therefore one in which the muscles last enumerated, those which approximate and render tense the vocal cords, are paralyzed. A *mixed* respiratory and phonatory paralysis is one in which both functions are impaired or lost. According to Bosworth, "it is quite a mistake to think that high tension of the vocal cords involves the necessity of their being stretched to their utmost by the arytenoid cartilage and the receding angle of the thyroid, thus turning their edges into absolute parallelism; more probably the tension of the vocal cords involves their edges being held in a state of firmness and rigidity, whether the chink be a straight line or an oval opening." *

Paralysis of the Inferior (Recurrent) Laryngeal Nerve.—*Varieties of Recurrent Laryngeal Paralysis.* Paralysis of the inferior or recurrent laryngeal is of more serious moment than that of the superior laryngeal, owing to its larger functions. As the result of an extensive lesion of the recurrent laryngeal nerve, all the muscles of the larynx, except the posterior cricothyroids, may be paralyzed, the picture being one of nearly complete unilateral or bilateral laryngeal paralysis. According to Exner and others, the muscles to which the inferior laryngeal nerve are distributed receive additional innervation from the superior laryngeal, which accounts for some of the unusual phenomena at times observed in laryngeal paralysis. A portion of the inferior laryngeal nerve has been resected without producing complete paralysis of the muscles which are usually regarded as receiving their entire nerve supply from the recurrent laryngeal nerve. Unilateral cases are chiefly caused by focal lesions, and bilateral cases by nuclear degeneration; although, in rare instances, even the complete bilateral type may be due to focal lesions, as when both sides of the median line of the postoblongata are involved, or when symmetrical lesions or extensive central lesions attack both recurrent laryngeal trunks. Partial forms of recurrent laryngeal paralysis are somewhat rarely observed, as unilateral or bilateral laryngeal abductor paralysis, or unilateral or bilateral laryngeal adductor paralysis, and paralysis of particular laryngeal muscles. Paralysis of the thyroarytenoid muscles (paralysis of the internal tensors) is a common affection, because these muscles are situated immediately beneath the mucous membrane of the surface of the vocal cords, and hence become implicated in inflammatory processes attacking them.

Bilateral Recurrent Laryngeal Paralysis. In bilateral recurrent

* An elaborate discussion of the anatomy and physiology of the larynx is not called for in a neurological work. It is only necessary to call attention in a general way to the motor mechanism of the larynx, and especially to its neural relations. For a discussion of the numerous problems connected with respiration and phonation, laryngological articles and treatises should be consulted.

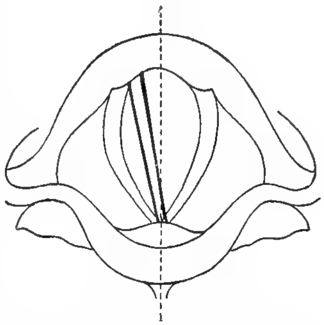
laryngeal paralysis the voice is lost (complete aphonia). Attempts to talk result in a strained whisper, and at every few words the patient is compelled to recover breath. Other symptoms may accompany those which have been mentioned, but they are collateral or secondary and are not essentially part of the paralysis. The patient, for instance, may have an irritative cough. This paralysis can be at once recognized on examination with the laryngoscope. The vocal cords are seen to be practically immovable, lying about midway between extreme abduction and extreme adduction, in the so-called "cadaveric position."

Unilateral Recurrent Laryngeal Paralysis. Recurrent laryngeal paralysis is most frequently unilateral, and most commonly due to pressure upon the nerve trunk. The patient suffers from weakness of the voice rather than from true hoarseness. In time the unaffected cord compensates to a greater or less extent for the one that is paralyzed, and little change in the voice is noted in ordinary conversation ; but the patient tires sooner in speaking, and the vocal range is lessened. In unilateral recurrent paralysis one cord is observed in the cadaveric position, while the other is placed normally and seems to respond normally to currents of air. The cord of the normal side sometimes passes across the median line to meet its crippled fellow. The rima glottidis becomes oblique, being deflected from before backward to the paralyzed side. In addition to the symptoms just mentioned, which are those that strictly belong to this form of laryngeal paralysis, are others, due to the same lesion as that which causes the paralysis of the larynx. As aneurism of the aorta is one of the most frequent of these lesions, the various symptoms of this affection sooner or later appear. Spasm of the larynx sometimes accompanies the paralysis when the lesion irritates as well as compresses or destroys the nerve. A number of cases have been recorded in which death has resulted from laryngeal suffocation brought on by paralysis of the recurrent laryngeal in cases of thoracic aneurism. An illustration of unilateral recurrent laryngeal paralysis, causing complete paralysis of the left vocal cord, is shown in Fig. 442. The case was one in which the paralysis was due to an aneurism of the transverse aorta. The patient had a brassy cough, and suffered from hoarseness amounting at times to almost complete extinction of the voice. (Stewart.)

Paralysis of the Posterior Cricoarytenoid Muscles (Laryngeal Abductor Paralysis). As the posterior cricoarytenoid muscles abduct the vocal cords at every expiration, paralysis of these muscles, if isolated, gives rise to one of the pure forms of laryngeal respiratory paralysis. Patients suffering from the bilateral form of abductor laryngeal paralysis have attacks of paroxysmal dyspnea, which become serious and more frequent as time progresses. Expiration and phonation are not affected, and attention is therefore not called to the

disease by any change in the patient's voice. The laryngoscope shows the vocal cords motionless, close to the median line (Fig. 443). Unilateral abductor paralysis is readily detected on laryngoscopic examination. While the cords are normal during expiration and phonation, during inspiration one cord is normally abducted and the other remains motionless. This form of paralysis can be mistaken only for one of the more complete types of recurrent laryngeal paralysis in which one of the cords assumes the cadaveric position. The chink of the glottis runs somewhat obliquely from before backward, while the arytenoid cartilage on the normal side passes in front of its fellow. Even when paralysis of the recurrent laryngeal nerve is eventually complete, the abductors frequently show a tendency to be first affected; indeed, it has been held that they are always first paralyzed, but this has been shown in several carefully studied cases not to be true.

FIG. 442.



Drawing of the larynx showing the position of the left vocal cord, as seen from above, due to pressure on the left recurrent laryngeal nerve by an aneurism of the transverse aorta. (Stewart and Major.)

Paralysis of the Lateral Cricoarytenoid Muscles (Laryngeal Adductor Paralysis). Bilateral laryngeal adductor paralysis is an extremely

FIG. 443.

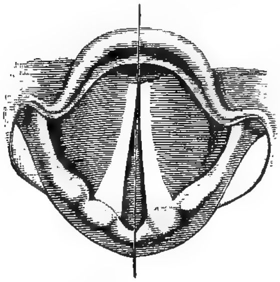


FIG. 444.

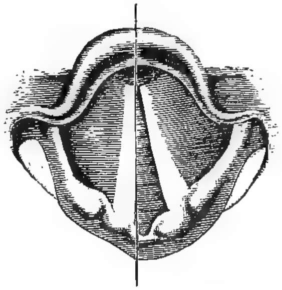
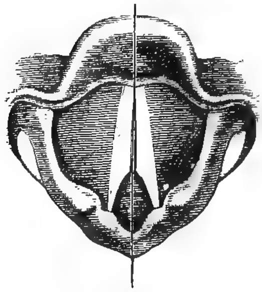


FIG. 443.—Bilateral abductor paralysis: position of the cords in deep inspiration. (Porcher.)
FIG. 444.—Unilateral adductor paralysis: position of the cords in attempted phonation. (Porcher.)

rare affection except as a part of complete recurrent laryngeal paralysis. Some of the recorded instances of this affection were more probably hysterical. The chief symptom is complete loss of the voice. According to Bosworth, this form of paralysis cannot be distinguished by laryngoscopic examination from complete recurrent laryngeal paralysis. While, theoretically, it would be supposed that the cords would recede from the middle line, they are probably ap-

proximated by the unparalyzed thyroarytenoids. Unilateral laryngeal adductor paralysis is extremely rare. The difference between the cadaveric position produced by unilateral recurrent laryngeal paralysis and the abduction which results from this form of paralysis is very slight. The only symptom is impairment of voice. The laryngoscope shows one of the cords extremely abducted. (Fig. 444.)

FIG. 445.



Paralysis of the arytenoideus muscle. (Porcher.)

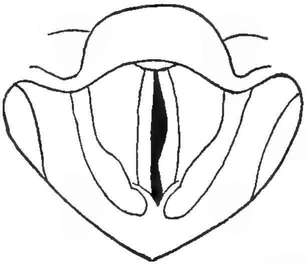
Paralysis of the Arytenoids.—The arytenoid muscle which passes from one arytenoid cartilage to the corresponding opposite cartilage is sometimes paralyzed, and especially by chronic inflammatory and catarrhal processes attacking the larynx. The voice may be greatly impaired or lost. The laryngoscopic appearances are shown in Fig. 445. In the posterior third of the glottis a triangular space is left, this not closing on phonation; but in the anterior two thirds of the glottis the cords are well approximated.

Paralysis of the Thyroarytenoid Muscles (Paralysis of the External Tensors).—This is a common form of laryngeal paralysis, due to lesions affecting directly the substance of the cords, the thyroarytenoid muscles lying just beneath the mucous membrane on the lower surface of the cord. It is a frequent cause of the hoarseness of voice which accompanies acute and chronic laryngitis. It may also be due directly to overuse of the voice. Clergymen, public speakers, auctioneers, and others who abuse their voices frequently suffer from this disorder. The voice is impaired especially in its purity and in its higher notes. The paralysis is generally bilateral, although occasionally it is unilateral. The rima glottidis is elliptical in shape.

Paralysis of the Superior Laryngeal Nerve.—Paralysis of the superior laryngeal nerve may be unilateral or bilateral, complete or incomplete. It is most frequently unilateral from a focal lesion at its origin or somewhere in its course, and when bilateral, like other bilateral paralytic affections, is more likely to be the result of nuclear degenerative disease. The cricothyroid muscles alone may be paralyzed, but this is exceedingly rare, and could scarcely be due to a trunkal lesion; more probably it would be the result of a partial degeneration of the nucleus of the superior laryngeal nerve on one or both sides. When the paralysis of the superior laryngeal nerve is complete, both the cricothyroid muscles and the thyroarytenoepiglottideus (thyroepiglottic) muscles show loss of function. The exact results of cricothyroid paralysis are not clearly known. The voice probably becomes hoarser and deeper than normal, while the

production of high notes is rendered difficult. This Gowers attributes to the fact that without the influence of the cricothyroid muscles the internal thyroarytenoids cannot act effectively, the latter being, of course, the most important phonatory muscles. In this respect the cricothyroid muscles may be regarded as accessory to the thyroarytenoids. Cricothyroid paralysis, when unilateral, can be recognized with the laryngoscope. The posterior portion of the glottis is displaced towards the paralyzed side, and the action of the epiglottis is impaired. The paralysis of the thyroepiglottic muscle allowing the epiglottis to be drawn forward, the glottis fails to be safely closed during deglutition, and food or drink passes into the larynx above the vocal cords. Another effect of lesion of the superior laryngeal is the production of anesthesia in the upper part of the larynx. In consequence of this anesthesia the foreign matter may not give rise to reflex coughing until some of it has passed below the vocal cords, and for this reason pneumonia may result. The arytenoid cartilages are movable during attempts at phonation, if the paralysis is confined to the superior laryngeal supply. The glottis has the appearance of a waving line on laryngoscopic examination. Bosworth says of paralysis of the superior laryngeal nerve that he does not know of any lesion which will produce the curious glottis observed when both superior laryngeal nerves are paralyzed,—namely, that in which the chink is divided by the approximation of the tips of the vocal processes. The appearance when the paralysis is unilateral is shown in Fig. 446.

FIG. 446.



Unilateral paralysis of the superior laryngeal nerve.

Laryngeal Paralysis associated with Hemiplegia.—A few cases of laryngeal paresis or paralysis apparently due to cortical lesion have been placed on record. When, however, the large number of hemiplegics and monoplegics is considered, these cases are comparatively rare, although it is possible, as has been suggested, that forms of laryngeal paresis are sometimes overlooked, especially if they are unilateral. A few years ago I had a considerable number of hemiplegics in the nervous wards of the Philadelphia Hospital examined laryngoscopically, and only three or four presented decided pathological conditions of the larynx of nervous origin. Simerka has carefully studied the subject of laryngeal paralysis in hemiplegia, reviewing the literature and summarizing a number of cases. He shows that there are a few well observed cases with autopsy of unilateral laryngeal paralysis from limited lesion of the cortex. These present fixation in extreme abduction of the vocal cord opposite

to the side of the lesion. Fixation of one vocal cord in the median line or in the cadaveric position has also been observed, probably a contraction due to cortical irritation. Simerka examined for Pierre Marie twenty-three hemiplegics who, on account of disturbance of speech or for some other reason, would seem to have had some laryngeal trouble. The time which had elapsed since the attack varied from one to eighteen years. In nineteen of these cases no involvement of the vocal cords was observed, and in four there was some disturbance of the right vocal cord Three of these patients had right-sided hemiplegia, the fourth left-sided hemiplegia.

Association of Laryngeal Paralyses with Tabes and other Degenerative Diseases.—Some of the most interesting cases of laryngeal paralyses and some about which our knowledge is most complete are those which occur in connection with tabes and other forms of chronic degenerative disease. Almost every variety of laryngeal paresis or paralysis has been observed in connection with tabes. Of thirty-three cases studied by Krause, thirteen showed gross functional disturbances, paresis and paralysis of all varieties coming and going with great irregularity. In some of these cases the motor disorder is an ataxia rather than a paralysis or a paresis. Besides tabes, laryngeal pareses of different types have been observed in cases of multiple sclerosis, and of course in progressive bulbar paralysis. Krause has pointed out the occurrence of unilateral or bilateral median positions of the vocal bands in some of these degenerative cases, and believes that they are in many instances produced by spastic contraction of the adductors rather than by paralysis of the abductors. This view of Krause is, however, not generally accepted; even he admits the occurrence of true abductor paralysis either central or peripheral, but believes that in some cases at least the laryngeal condition is due to spasmodic contraction of the adductors. Others have shown that in the laryngeal paralyses associated with tabes and other degenerative diseases paralysis of the abductors is likely to occur first, and after this paralysis of the internal tensors.

Association of Laryngeal Disorders with Functional Neuroses.—Krause found various forms of laryngeal paralysis associated with certain well known functional neuroses, such as chorea, hysteria, hysteroepilepsy, and railway spine. In chorea he observed tremulous action of feebly distended vocal bands and paresis of the abductors in almost all instances. In no case were true choreic movements exhibited by the laryngeal muscles. In cases of hysteria and hysteroepilepsy, hemianesthesia of the mucous membrane of the nose, soft palate, pharynx, and trachea, mostly on the left side, was found, with hemianesthesia of the larynx, and in one instance there was perverse action of the vocal bands, consisting of approximation on inspiration and widest separation during expiration and phonation. In two cases of railway spine with widespread cuta-

neous anesthesia there was anesthesia of the mucous membranes of the upper air passages, except at a few irregular spots.

Etiology and Pathogenesis.—In the discussion of etiology the laryngeal paralyses can be considered together. These affections may be due to focal lesion at the origin or in the course of the vagus or of its laryngeal branches. Any lesion attacking these structures may cause the affection. Among the most frequent lesions are enlargement of the thyroid body, of the glands of the neck or of the bronchial glands, mediastinal or other intrathoracic tumors, esophageal growths, pleuritic adhesions, and fibroid induration of the apices of the lungs. One of the most frequent causes is aneurism of the aorta, or of the carotid, innominate, or subclavian artery. Hypertrophy of the heart or large pericardial effusions may, by displacing the heart, cause pressure on the recurrent laryngeal nerve. The left recurrent laryngeal nerve is especially exposed to pressure from aneurisms, mediastinal tumors, and enlarged bronchial glands. The nerve of the right side is more likely to be included in fibroid induration of the apices of the lungs or in pleuritic adhesions. Gunshot or other injuries to the nerves, and surgical injuries to the nerves such as are produced by operations on the neck, are other causes. Tumors, meningitis, aneurisms, and hemorrhages at the base of the brain are other causative lesions. In a case reported by Daland, in addition to an unusual lesion of the internal capsule (the cicatrix of an old hemorrhage) a small aneurism of the right vertebral artery was found, which from its position might have pressed upon some of the root bundles of the vagus. In a case referred to by Krause of gliosarcoma of the striatum and left temporal lobe there was paralysis of the right half of the larynx. This case confirms the existence of a cerebral centre for the laryngeal muscles. Any form of laryngeal paralysis may be due to an infectious or toxic cause. Such paralysis is frequently seen during diphtheria, or during the convalescence from this disease. Like other nerves, the recurrent laryngeals may be attacked with neuritis, the result of exposure to cold or of the influence of agents acting upon the blood. In one case of spinal syphilis Krause found the left vocal band immobile near the middle line, but it became active again in connection with return of function in the left extremities under treatment by inunction. In six cases of plumbic paralysis no laryngeal manifestations were noted, but in one case there was paralysis of the left vocal band, and in another there were conspicuous tremulous movements of the vocal bands with adductor paralysis. Lublinski has reported on six cases of posttyphoid laryngeal paralysis observed by him. In one severe case paralysis came on in the fourth week; in the others after defervescence, as in most cases previously reported. In one case a right sided recurrent paralysis followed pleuropneumonia. Usually the condition comes on in convalescence, with inspiratory dyspnea, but

without alteration of the voice. In one case of paralysis of the recurrent nerve the soft palate was also paralyzed. Cases of paralysis of the vocal cords following influenza have been reported. Forms of functional paralyses are sometimes due to excessive speaking or other abuse of the voice.

Pathological Anatomy.—The pathological anatomy of laryngeal paralyses has been almost sufficiently considered in the last paragraph when referring to their most frequent causative lesions. A few investigations have been made of the muscles, nerves, and nerve centres. In an interesting case reported by Onodi, of bilateral aneurism of the aorta, in which the right vocal band was immobilized in the cadaveric position, while that of the left side was practically in the middle line, section showed that the right recurrent nerve was injured in its totality by the aneurism of the left side. Onodi succeeded in preparing the muscles, isolated, with their nerves detached, and submitted them to microscopic examination. He found a degenerescence of all the muscles and nerves on the right side, while on the left the most serious alterations had implicated the posterior cricoarytenoid muscle and its nerves, the internal thyroarytenoid muscle being less implicated, and the lateral and transverse to a still less extent. In 1886 Cohen presented to the American Laryngological Association the larynx and some microscopic sections of nerve tissue from a case of bilateral paralysis of the posterior cricoarytenoid muscles which he had exhibited in 1879. The larynx showed the fixed, spastic position of the vocal bands in the middle line that they had occupied for nearly ten years during life. The posterior cricoarytenoid muscle of the left side was quite atrophied, that of the right side much less, but the bundle of fibres of the arytenoid going from the base of the left cartilage to the top of the right was equally atrophied, and all the remaining muscles were apparently normal. The right recurrent laryngeal nerve, near the point where it leaves the pneumogastric, showed marked atrophy in one half of its area; the left nerve exhibited no pathological change. A small triangular cavity, from two to four millimetres in extent, was found in the outer section of the lenticula.

Diagnosis.—The diagnosis of the existence of a laryngeal paralysis, and also of its special type, is finally made with the laryngoscope. The existence of some of the forms of laryngeal paralysis is often unsuspected for a long time. This is particularly true of partial unilateral palsies either of the complete recurrent type or of one of the recurrent subtypes. The patient suffers perhaps from occasional shortness of breath and slight difficulty in talking, or is easily tired by conversation, but is not aware of the real nature of his trouble until an aggravation of his laryngeal symptoms or the awaking of new symptoms leads to a laryngoscopic examination. The patient may, for example, suffer from deep-seated localized pain and have

unilateral dilatation of the pupil, and because of these attention will be more strongly directed to the partially latent or overshadowed laryngeal symptoms. The laryngoscope is used, and a unilateral paresis or paralysis of the vocal bands is discovered. The focal diagnosis of laryngeal paralysis—the fixing of the site of the lesion causing it—is usually to be made by a consideration of the associated clinical phenomena. Care needs to be taken not to confound a true neural paralysis of the laryngeal muscles with a paralysis or pseudoparalysis due to inflammatory or other pathological processes in the larynx. Hooper has called attention to the fact that anatomical rather than neural factors are often present to produce the abnormal positions of the vocal bands frequently attributed to paralysis, and especially to paralysis of individual muscles. These factors are the relative sizes and other peculiarities in the structures of the larynx.

Prognosis.—The prognosis of laryngeal paralyses varies with their causes and type. Some forms of focal lesions causing them, as tumors, glandular enlargements, or pleuritic adhesions, may improve under medicinal or surgical treatment, or may be of such a character as not to permit of any improvement. A neuritis may be cured, although the forms of neuritis which attack the vagus and its branches are among the most serious and fatal. With regard to aneurism of the arch of the aorta and the other large bloodvessels of the thorax, it need only be said that the prognosis is that of the aneurism itself; but occasionally in these cases the pressure is for a time partially relieved by treatment or by the particular course which the disease may take so far as relates to the production of pressure and displacement. Cases associated with tabes and other forms of progressive degenerative diseases are of course of unfavorable prognosis, although they often make very slow headway, and sometimes, like the other symptoms of these affections, remain at a standstill for a considerable period. The forms of paralysis with most favorable prognosis are the hysterical, and those which are due to infectious and toxic processes: diphtheritic laryngeal paralysis, for instance, has a comparatively favorable prognosis. One important practical point of prognosis should not be forgotten, namely, that in some of the severe types of laryngeal paralysis the patient's life may be suddenly endangered. In paralysis of the superior laryngeal, for example, as has been shown, food or drink getting into the anesthetic upper chamber of the larynx may be carried downward into the trachea or bronchi before reflex irritation is excited, and pneumonia or bronchopneumonia may be the result. A long time is required for the regeneration and reuniting of the laryngeal as of other nerves which have been severed in surgical operations or as the result of injuries. The time set by some authorities, based upon recorded cases in which such union has taken place, has been about a year or a little more. In cases of peripheral paralysis

after the muscles have completely atrophied, even although partial restoration of the nerve takes place, the muscles may not regain their bulk and functions. The importance of prompt action in some cases where death is threatened is well illustrated in a case reported by Hartwig of bilateral abductor paralysis. When the patient came under observation he was suffering from labored respiration and well marked inspiratory stridor on exertion. Immediate tracheotomy was urged, but was put off by the patient. On the fourth day after his admission to the hospital the man was seized with a paroxysm of dyspnea, and death seemed imminent, but was averted by tracheotomy.

Treatment.—The treatment of laryngeal paralyses must depend upon their cause. In a few instances tumors and glandular enlargements can be removed by the surgeon; much more frequently they are malignant, or are so extensive, infiltrating, and deep-seated as to make an entirely successful operation impossible. When the palsies are postinfectious or toxic, great benefit will be derived from the administration of strychnine, either by the mouth or hypodermatically, and especially by the latter method. As good results seem to be achieved when the injections are made in parts more or less remote from the larynx as when made in its vicinity. Insufflation of salts of strychnine into the larynx has been practised, but the method is one not to be commended. When dependent upon aneurism, induration of the larynx, enlargement of the heart, or other formidable organic lesion, little of direct benefit can be secured by treatment. No treatment is of curative value in the cases associated with neuraxial degeneration. Electrical treatment of laryngeal paralyses has long been practised, and in particular cases has much to commend it. The treatment may be either extralaryngeal or intralaryngeal. When the former method is employed, one or both of the electrodes are applied as closely as possible to the parts of the larynx affected. When the faradic battery is used, the current may be of considerable strength. The galvanic battery interrupted at intervals may also be used, but it should be remembered that applications of galvanism to the neck are not unassociated with danger, owing to the effect upon the pneumogastric. Strong currents and currents abruptly interrupted should not be carelessly used, as syncope may be produced. Intralaryngeal applications should be resorted to only by those who have some expertness in manipulation, but when used by skilful operators they sometimes result in marked temporary, and in rare cases more or less permanent, improvement. A laryngeal electrode is introduced into the larynx with the assistance of the laryngoscopic mirror, and a weak current applied. Electrical treatment is of special value in hysterical palsies of the vocal cords: a single application will sometimes restore the voice. Vocal gymnastics may also be employed, especially in functional cases.

When infectious cases are on the road to recovery, a favorable result may be hastened by using electrical treatment in addition to the administration of strychnine. When suffocation is threatened, it may be necessary to resort promptly to tracheotomy, as in the case of Hartwig, to which reference has been made under prognosis. As the threatened fatal result is often due to transient causes, such treatment may prove successful and prolong the patient's life for an indefinite period. When paralysis is due to neuritis, the internal treatment ordinarily applicable to neuritis should be resorted to, but with special care, as such remedies as the salicylic compounds, the iodides, and the bromides, which are useful in so many forms of neuritis, may have unusually depressing effects in laryngeal cases. The depressing results of these drugs can in a measure be counteracted by conjoining the use of strychnine with the special antineuritic remedies. In syphilitic cases the use of mercury by inunction or hypodermatically should be resorted to; or iodides by the methods employed in other cases of syphilis. When laryngeal paralysis is a part of the syndrome of bulbar paralysis, and is associated with dysphagia and other serious symptoms, it may be necessary to resort to artificial feeding with tubes through the nose or the mouth in order to prolong the life of the patient.

SPASM OF THE LARYNGEAL MUSCLES.

Varieties and Clinical History.—Spasm of the larynx is rare as an isolated affection, although comparatively common, both in children and in adults, as a reflex phenomenon in various forms of disease of the respiratory apparatus. It may be *acute* or *chronic.* When acute it usually presents itself as a paroxysmal affection in the course of some other disease, such as whooping cough, laryngeal tuberculosis, or syphilitic or other inflammatory disease of the vocal apparatus. Spasm of the larynx is most frequently *bilateral*, but may in rare instances be *unilateral*, in these cases being, as a rule, due to the direct irritation of either the superior or the inferior laryngeal nerve. Even when the irritation is unilateral, the effects in some instances may be exhibited on the muscles of both sides. Spasm of the larynx may be of any of the groups of muscles, but adductor spasm is the most frequent form. The adductors, being physiologically the most powerful of the laryngeal muscles, are most likely to be strongly brought into play in such actions as coughing, and hence are most liable to spasm as the result of direct or indirect irritation. Laryngeal spasm may be treated of as it occurs in adults or in the special types as it occurs in children. One of the most common varieties of laryngeal spasm in children is the so-called *laryngismus stridulus.* Spasm of the glottis as it occurs in adults is in most instances reflex in character. "Laryngeal spasm may be excited by the entrance of food, drink, or other foreign

substances into the larynx; by irritating topical applications, as by means of the sponge or probang; and by the presence of movable tumors. It also occurs in connection with tuberculosis and syphilitic disease of this organ. In the latter cases it is altogether probable that the spasm is excited by the entrance of solid or liquid food into the cavity, the act of deglutition being seriously interfered with by these affections. In the same way, any affection which interferes with this act may be attended with laryngeal spasm, such as pharyngeal paralysis, ulcerative processes in the pharynx or esophagus, as well as tumefaction in any portion of the fauces. In these cases the laryngeal spasm becomes a grave symptom according to the extent to which the food is thus diverted into the air tract. Aside from the above cases, the disease probably is largely confined to those instances in which the muscular contraction is a reflex phenomenon excited by some diseased condition either in the larynx or in some other portion of the upper air tract." (Bosworth.) Oppenheim has recorded a case of a peculiar form of spasm of the larynx due to focal lesion. A tumor of the cerebellum compressed the pons and oblongata. The roots of the vagus and accessorius were hyperemic, flattened, and atrophic. During life there had been trembling of the head and of the upper extremities on voluntary movements only. But there had been a continuous rhythmic tremor of the soft palate, as well as of the outer and inner musculature of the larynx. The larynx was continually drawn up and lowered. The rhythmic contraction of the cricothyroid muscle could be felt externally; and on laryngoscopic inspection continuous trembling of the inner laryngeal muscles and the movements of the arytenoid cartilages could be seen. These manifestations, which produced disturbances in deglutition, as well as in speech and voice, had been observed in varying intensity for a period of about two months. A case has been recorded by Baginsky of clonic rhythmic spasm of the abductor muscles of the larynx, the rate of the movements being from fifty to fifty-four per minute. The movements were constant, almost regular clonic spasm of the vocal bands and arytenoid cartilages recurring. The short abduction movements brought the vocal bands to about the cadaveric position, but no farther. They followed rather regularly at the close of the expiratory movement, and were in part associated with it, but they did not extend into the inspiratory movement. Rapid respiration and voluntary inspiratory stridor arrested the clonic spasm for a few minutes, after which it recurred with greater intensity. Baginsky suggests the term nystagmus of the vocal bands, the manifestations being analogous to nystagmus of the eyes. The spasm was observed in an hysterical patient sixty years old. The patient had a history of hysterical manifestations extending back for more than forty years. This included recurrent hemorrhages, paralysis and anesthesia of

the extremities, aphonia, paroxysmal dyspnea, spasmodic cough, frequent emesis, convulsion, photophobia, and double blepharospasm. Furundarena Labat records an unusual case of laryngeal spasm under the name of chorea of the larynx. The patient, a woman twenty years of age, had had an attack of the grippe, after which she had in a great measure lost her voice and was subject to a peculiar dry cough of an especially irritating character, which continued during the day but was entirely absent during the time of sleep. Examination with the laryngoscope revealed most curious, sudden, rapid, and irregular contractions of the inferior vocal cords. These peculiar clonic convulsiform movements led to a diagnosis of laryngeal chorea. Suspecting that the cause of the laryngeal neurosis might be of reflex origin from disease of the nasal mucous membrane, the Schneiderian membrane was touched with a solution of muriate of cocaine, and it was found that during the time of action of this drug the clonic contractions of the larynx ceased as if by enchantment. On the contrary, when an application of a small quantity of the caustic paste of Mackenzie was made to the hypertrophied mucous membrane of the nasal fossæ there was so great a reaction produced in the larynx that the patient passed three or four days of misery, respiration being impeded and the dyspnea being greatly increased. After complete removal of the hypertrophied membrane by means of the galvanocautery, the symptoms disappeared and the cure was complete.

Etiology and Pathogenesis.—Numerous experiments long since demonstrated that if the superior laryngeal nerve be divided and its central end be stimulated, bilateral spasm of the glottis will follow. In these cases when the stimulation is sufficiently strong the impression is conveyed to the laryngeal centre in the postoblongata and is thence irradiated to the centres of the other side, the resulting spasmodic phenomena implicating the muscles supplied by both the superior and inferior laryngeal nerves. When the recurrent laryngeal is cut and its efferent fibres are stimulated, the spasm is usually confined to the muscles supplied by this nerve on one side, unilateral abduction of the vocal cords occurring. Pressure or irritation of the roots of the vagus or the trunk of the vagus before the laryngeal nerves are given off may lead either to unilateral or bilateral spasm or to paralysis of one side and spasm of the other. Serious or even fatal obstruction of the larynx may in this way ensue. Anything producing marked irritation of sensory nerves may cause this irritation to be reflected to the laryngeal centres and nerves, and it is for this reason that acute laryngeal spasm is so frequently met with in children during dentition or when suffering from affections of the respiratory or the gastrointestinal tract. Its frequent occurrence in whooping cough is well known, and, although commonly recovered from, is not unattended in special instances with

great danger. Among the exciting causes of laryngeal spasm in the adult are the introduction into the larynx of any substance capable of producing irritation, as the accidental admission of food or drink into the glottis, the inhalation of irritating vapors, the passage of a probe into the larynx, or any irritating substance used for topical applications in the treatment of catarrhal or other affections of the throat and larynx. In syphilitic and tuberculous disease of the larynx, and in diphtheria, laryngeal spasm is sometimes caused by the interference with deglutition, this permitting the passage of irritating foreign substances into the larynx and trachea. Various bulbar affections and various diseases of the last four cranial nerves, whether bulbar, cerebral, or peripheral, may in like manner indirectly be the cause of laryngeal spasm. The faucial and pharyngeal paralysis present in these cases interferes with the act of swallowing, and allows the passage of foreign substances into the laryngeal tract. Paroxysms of epilepsy and eclampsia are not infrequently induced by attacks of spasm of the larynx, pharynx, and masticatory muscles. This being the case, it is not improbable that in rare instances spasm confined to the laryngeal muscles is of cortical origin.

Diagnosis, Prognosis, and Treatment.—The diagnosis of the different forms of laryngeal spasm is to be made by a close study of the etiology and symptomatology of the affection in the lines of the statements already made. Both in children and in adults spasm of the larynx may be sometimes confounded with laryngitis, especially the subglottic form; indeed, in this affection some spasm may be present. It is necessary, however, to distinguish between a disorder purely neural and such an inflammation. The presence of fever, of croupy cough, and of secretion from the mucous membrane of the larynx will assist in the diagnosis. The discovery of sources of reflex irritation in the nose, throat, pharynx, gums, teeth, or upper air passages will be of assistance. The prognosis of laryngeal spasm is dependent upon the nature, severity, and extent of the lesion causing it. While in many cases serious, it is, on the whole, more favorable than that of the marked types of laryngeal paralysis, although some cases of laryngeal spasm in children end fatally. The condition of the general health of the patient has an important bearing upon prognosis. Treatment should be directed to the cause. When laryngeal spasm is associated with serious focal lesions at the origin or in the course of the laryngeal nerves, when it is one of the phenomena of progressive degeneration, or when it is associated with malignant or serious disease of the respiratory channels, little can be done in the way of treatment, although surgical procedures and the administration of absorbent remedies may sometimes accomplish beneficial results. Diseases of the respiratory passages which are amenable to treatment should receive close attention.

RESPIRATORY DISTURBANCES DUE TO DISEASE OF THE VAGUS.

The Nervous Mechanism of Respiration.—The nerve supply to the trachea and bronchi is directly from the pneumogastric (recurrent laryngeal) and the gangliated system; that to the substance of the lungs is from the anterior and posterior pulmonary plexuses, which are formed chiefly by the union of numerous branches from the pneumogastric and from the gangliated system. The neurosal affections of the lungs are therefore due to lesions and disturbances of the vagus or of the "sympathetic." The fibres from the sympathetic to the lungs and bronchi are chiefly vasomotor; and the pulmonary sympathetic neuroses are therefore probably vasomotor affections. The pneumogastric mechanism is by no means the only one whose disarrangement leads to respiratory disorders which can be properly regarded as of nervous origin. Besides the vagal centres in the oblongata, other respiratory nerve centres are probably situated in the cortex of the brain, and certainly in the spinal cord. The spinal accessory, phrenic, and thoracic nerves are important accessory nerves of respiration, and their nuclei are therefore in this sense respiratory centres, lesion or disturbance of which may lead to various disorders of breathing. The conducting portion of the respiratory apparatus is also of great importance, and includes an extensive series of fibres, as follows: (1) afferent fibres from the surface of the body which generally influence the respiratory cortical centres; (2) afferent fibres from certain parts of the body which influence the respiratory centre in the oblongata; (3) affero-accelerating fibres which are contained in the trunk of the vagus and go to the respiratory centre; (4) affero-inhibitory fibres to the respiratory centre which pass in the superior and inferior laryngeal nerves, and probably, also, in the splanchnic nerves; (5) efferent fibres which connect the cortical motor centres with the respiratory centres in the oblongata, and probably also directly with the spinal centres; (6) tracts which connect the respiratory centre in the oblongata with the spinal centres; (7) connections of the spinal nuclei with the muscles of respiration by the thoracic, phrenic, and spinal accessory nerves, and by the nasal branches of the facial, and some of the fibres of the superior laryngeal nerve. (Ross.) Space will not permit us to discuss in detail the effects of lesions of different portions of this complicated mechanism. These effects are, however, easily understood from this physiological summary.

Varieties of Respiratory Neuroses.—The respiratory affections due to lesions and disturbances of the vagus may be classified as *tachypnea*, abnormal frequency of breathing; *brachypnea* (or *spanopnea*), abnormal slowness of breathing, or shortness of breath; and *respiratory arhythmia*, in which the breathing is irregular. The term

dyspnea, or difficult breathing, is applied without much precision to almost any form of rapid, slow, or irregular breathing; *apnea* properly refers to that condition in which the blood is unduly oxygenized, an excess of oxygen in the blood preventing proper stimulation of the respiratory centre in the bulb; and *orthopnea* is a condition of difficult and labored, often rapid breathing, from which the patient can get relief only by assuming the upright position. *Asphyxia* is a condition which is caused by arrest of respiratory movements owing to deficiency of oxygen in the blood which impairs or destroys the excitability of the respiratory motor centre in the oblongata. *Cheyne-Stokes respiration* is a peculiar form of breathing with a gradually ascending and descending rhythm and a periodical pause, which is usually explained on the theory that when the excitability of the respiratory centre is greatly diminished the blood must become surcharged with carbonic acid in order to excite it, "the increased inspiratory efforts thus induced diminish the venous state of the blood, and the respiration becomes less powerful, until it is finally arrested for a time until the blood becomes again surcharged with carbonic acid." (Ross.) It will be seen that all these forms of respiratory disturbance might be properly included under the three heads of tachypnea, brachypnea, and respiratory arhythmia.

Tachypnea.—Strübing has reported a number of interesting cases both of tachypnea and of brachypnea (spanopnea). In tachypnea the respiratory movements are greatly increased in frequency and diminished in depth. In one of two cases the attacks ceased after the restoration of a retroflexed uterus. In another case the patient suffered from extreme nervousness and mental deficiency. In this connection the cases of so-called hysterical asthma or hysterical breathing must be borne in mind. These, indeed, may perhaps be regarded as due to functional disturbance of the vagus, although they must be separated from cases of tachypnea due directly to lesion of the pneumogastric or to special reflex causes. Cases of this kind in which the patients have a series of rapid and forced respirations have fallen under my observation. Great excitement usually attends these attacks of rapid breathing, the patients often fearing a fatal termination. Coates has reported several cases in which patients with tachypnea of nervous origin were supposed to be suffering from phthisis, but in which careful examination showed no organic lesion of the lungs. A few cases in which dyspnea was caused by a lesion directly affecting the vagus have been put on record.

Brachypnea.—In two of Strübing's cases the patient suffered from attacks of slow and deep breathing. In one of these cases the paroxysms were so severe as to amount to orthopnea with subjective dyspnea. In another case the attack seemed to be due to irritation of the inspiratory inhibitory fibres of the superior laryngeal

nerve during the act of swallowing. In a third case it appeared as asthma, and seemed to be excited reflexly by irritation of the trigeminal fibres in a case of chronic rhinitis with hypertrophy of the turbinated bone. The irritation was probably reflected in this case from the trigeminus to the pneumogastric centres.

Nervous Asthma.—Some forms of so-called nervous asthma may certainly be regarded as neuroses of the vagus. In consequence of disturbed innervation of this nerve, tonic spasm is supposed to take place in the circular muscles of the bronchi, and especially in those of finer calibre. In this way an emphysema is produced. This bronchial spasm forms an impediment much more easily overcome by inspiration than by expiration, and hence the disturbances of breathing are more marked during expiration. (Hirt.) Mays, who regards asthma, in one of its most usual forms at least, as certainly a spasmodic neurosis of the pneumogastric, recommends the use of hypodermatic injections of strychnine and atropine in its treatment. He begins with one fiftieth of a grain of strychnine and the one hundred and fiftieth of a grain of atropine daily, gradually increasing the former to one twenty-fifth or one twentieth of a grain and the latter to the one hundredth of a grain. After a thorough impression is made on the disease the drugs are administered every other day, and as the patient improves are gradually abandoned. Measures to control the cause of the attacks and to build up the general system should also be adopted. Another treatment of spasmodic asthma which has much in its favor is galvanization of the pneumogastric. For the treatment of the attacks Hirt recommends pyridin, which was first suggested by Sée. Inhalations of amyl nitrite and the administration of one of the preparations of lobelia are also efficient at times in giving relief.

Lesions of the Pneumogastric and Pulmonary Consumption.—According to Mays, disease of the vagus is the cause of pulmonary consumption. Whether his views are accepted or not (and I am not inclined to accept them), the facts which he has collected are of great interest. He has tabulated a very large number of cases with pulmonary disease, in which the vagus of one side or both vagi were affected by disease or injury. His tables show that lesion of the vagus was undoubtedly associated with disease of the lungs in nearly all the cases. With the addition of cases investigated by a few other observers, he presents definite records of one hundred and four cases of phthisis, fifty-four cases of pneumonia, and fifteen cases of other forms of lung disease, in which the oblongata or vagi were disorganized. In favor of his view he argues that both alcohol and syphilis, on account of their destructive affinity for the nervous system in general and for the pneumogastric nerves in particular, have the power of inducing this disease. "The testimony which has been collected," he says, "thus far lends no encouragement to the belief, then, that

pulmonary consumption is a disease which is created by a specific virus, but rather that it is a condition which evolves slowly out of preexisting pulmonary disorders. For it is manifest that in every one of the tabulated histories of cases given in the previous pages the nature of the lung disease depended on the acuteness or the chronicity of the vagus lesion. Division, injury, or acute disease of the vagi always results in œdema, hyperæmia, hæmorrhage, or bronchitis, but never in phthisis,—the last disease only being produced when the vagi were subject to a slow process of devitalization, such as would take place from long continued pressure or protracted disease. From these data one can legitimately infer that all the morbid phenomena occurring in the lungs are but different steps in the same process which may primarily begin in a simple œdema or catarrh and terminate in pulmonary phthisis."

CARDIAC DISTURBANCES DUE TO DISEASE OF THE VAGUS.

Physiology of the Cardiac Nerves.—If the depressor nerve of the heart is divided, and the proximal end stimulated with a faradic current, a gradual but marked fall of pressure in the carotid artery occurs, and if the end connected with the heart is electrically stimulated the beat of the heart is retarded, and its action may even be stopped in diastole. After division of both pneumogastric nerves the beat of the heart may be quickened by stimulating the cervical spinal cord, thus acting upon the so-called accelerator nerves. Disorders of cardiac movement may depend upon disease or disturbance of either the depressor or the accelerator nerve, or of inhibitory nerves distinct from the depressor although like it derived from the vagus. Stimulation of the depressor nerve causes pain, proving that this is a sensory nerve to the heart.

Tachycardia.—Tachycardia is a persistent, rapid beating of the heart. It may be a part of the syndrome, or, in rare instances, the only symptom of vagus lesion or disturbance. Attacks of tachycardia may last for a period varying from a few seconds or minutes to several hours. Frequently the paroxysms are initiated by flushing or throbbing. The pulse may reach as high as two hundred beats, although commonly it does not rise to more than one hundred and fifty. The patient suffers from intense anxiety, and may experience nausea and be excessively prostrated. Although the pulse is very rapid, the heart beat may be weak. In four cases reported by Kelly the essential feature of each case was the tendency to paroxysmally recurring attacks of extraordinary rapidity of the heart's action without sufficient cause. Kelly, after summarizing and comparing the views of a considerable number of observers, concludes that the majority of cases of *essential paroxysmal tachycardia* are due to a transitory paresis of the vagus, and that the seat of the disturbance is in the oblongata.

In the series of cases of pneumogastric disease tabulated by Mays are four of tachycardia. Kelly attempts to explain essential paroxysmal tachycardia by the neuron theory * suggested by Dercum and others in explanation of the phenomena of hysteria, hypnotism, and allied conditions. Sudden and excessive demand upon the functional capacity of the nerve cells of the vagus nucleus, or the slow action of fatigue or defective nutrition, may, according to this view, cause excessive neuronal motility, and, this leading to unusual retraction of the cell processes and the consequent breaking of the nerve circuit, the heart is no longer properly controlled. The diagnosis of essential paroxysmal tachycardia must be made by excluding all cases of permanent tachycardia due to known lesions, and also those cases which arise reflexly. The best permanent effect may be anticipated from the avoidance of excessive exertion and from paying particular regard to the general nutrition and welfare of the body. As tachycardia is one of the most important symptoms of exophthalmic goitre, attention is naturally turned to the vagus in attempting to arrive at a satisfactory explanation of this disease. Hirt includes exophthalmic goitre under diseases of the vagus, although a careful reading of his article shows that he seems to be uncertain whether it should be so regarded. Sattler proposed to assume in exophthalmic goitre a circumscribed lesion in the region of the vagus centre, by which the inhibitory action on the heart is diminished or suspended; but, as remarked by Hirt, he does not consider that the absence of other vagus symptoms can be held to disprove this, as this inhibitory action actually can be suspended and the other functions of the nerve remain intact. The symptoms of Graves's disease have been produced by cutting the restiform body. According to Putnam, even the results of thyreoidectomy (and, it might be added, of the internal thyreoid treatment) do not necessarily prove that the goitre is the cause of the other symptoms. "The thing which is needed for a cure in

* As this book approaches completion new views regarding nerve cells are announced. These views, which are the outcome of Apáthy's studies on the nervous systems of the leech, earthworm, rabbit, etc., were presented by Thomas H. Montgomery, Jr., Ph.D., at the meeting of the Philadelphia Neurological Society, October 25, 1897, and will be published in the Journal of Nervous and Mental Disease. According to Apáthy, the nerve cell is the producer of neurofibrils, while the ganglion cell produces the force which is to be conducted. Some of the neurogliar cells, of the leech at least, produce neurofibrils, some neurogliar fibrils, and some both kinds of fibrils. A neurofibril, which arises in a nerve cell, passes out of one of its processes, and may then traverse several ganglion cells, and finally end in or around a muscle or sense cell. The neurofibrils take part in the network of the cells, and none terminate or arise within the ganglion cells. The cell process of a ganglion cell contains both cellulipetal and cellulifugal neurofibrils. Apáthy speaks also of direct anastomosis of nerve processes. Should these teachings be accepted, the present conception of the "neuron" will require to be abandoned or greatly modified.

every case, and in every stage of the disease, is an adequate physiological rest for the disturbed nerve centres, and any influence may secure this which removes a good number of peripheral stimuli, or any influence which increases the stability of the central nervous system. That the diseased and engorged thyreoid may be a centre for these abnormal stimuli cannot be doubted." As bearing upon this question of the pneumogastric origin of exophthalmic goitre, the theory recently advanced by Hale White that the recurrent laryngeal nerve has trophic functions for the thyreoid gland is of interest. In two cases of recurrent laryngeal paralysis from aneurism of the aorta marked atrophy of this gland occurred. It is, however, not my intention to discuss exophthalmic goitre at length, but simply in completion of the survey of the affections of the vagus to direct attention to some of the views which seem to support the theory that it is either primarily or secondarily a disease of the vagus. The most reasonable view of the pathology of this disease would seem to be that which regards it as of glandular origin,—in other words, that the nervous as well as other symptoms are the outcome of the perversion of the functions of the thyreoid gland; but this is by no means settled.

Brachycardia.—Brachycardia (or bradycardia) is an affection in which the beat of the heart is slowed. In extreme cases the beat may fall to ten strokes or less. The most important accompanying symptoms are those which result from the abnormal manner in which the blood is distributed. Brachycardia is frequently paroxysmal, although a persistent slow pulse is not unusual. General weakness, impairment or loss of consciousness, syncope, and even convulsions, are sometimes associated symptoms. Senile epilepsy is said to be a not infrequent result of paroxysms of brachycardia in the aged suffering from arterial sclerosis. Brachycardia, like tachycardia, may be of an essential paroxysmal type, or it may be symptomatic. The symptomatic form has been noted in numerous diseases of the nervous system, such as encephalic tumor, meningitis, or hemorrhage, and disseminated sclerosis. Organic disease of the heart or blood-vessels is another comparatively common cause, and it may be due to toxic or infectious agents acting upon the pneumogastric nerve or its centres. Exhaustion and toxemia play the most important rôle in its causation, in all probability acting through the vagus, although the exact mechanism of its production cannot be given. An attack of brachycardia has terminated in death and the most painstaking autopsy has failed to detect a lesion. The diagnosis of the existence of brachycardia is, of course, not difficult. It is made simply by a careful examination of the pulse. So far as the paroxysms are concerned, the prognosis of brachycardia is probably somewhat more serious than that of the more distressing tachycardia. Atropine in decided doses should be employed if good reason exists for referring

the brachycardia to irritation of the vagus; otherwise the treatment should be symptomatic.

Cardiac Arhythmia.—Certain peculiarities of cardiac rhythm due to paresis or other disorder of the vagus are classed under the head of cardiac arhythmia. The arhythmia is determined both by studying the heart directly and by examination of the pulse. Among the varieties of cardiac arhythmia indicated by the pulse are those characterized by the deficient, the intermittent, and the alternating pulse. A beat may be wanting either because the heart fails to contract fully or because the beat is not transmitted from the heart to the point where the pulse is taken. The alternating pulse is one in which its volume varies from beat to beat, although no distinct loss of beat occurs. Sometimes the heart beats are separated by intervals of unusual length. Finally, the heart, because of faulty innervation, may exhibit so great an irregularity in frequency, and so much violence in its action, that the disordered movement may deserve the name of *delirium cordis.* Many forms of cardiac arhythmia are, of course, dependent upon organic disease of the heart or of other organs. Among affections which cause symptomatic cardiac arhythmia are atheroma of the coronary arteries, valvular diseases, fibroid myocarditis, and all other diseases which directly or indirectly produce malnutrition of the heart; while among the causes which act more directly upon the pneumogastric are toxic and infectious agents. The pneumogastric is also directly or reflexly affected by disorders of digestion, by sudden blows, and by violent emotion. A focal lesion, such as a tumor, hemorrhage, or area of softening, may affect the vagal centres or the trunk of the nerve and thus cause arhythmia. A pulse which loses a beat at irregular intervals is frequently observed in neurotic patients who have gouty or lithemic tendencies.

Irritable Heart.—Under the name of "irritable heart" Da Costa has described an affection in which the main symptoms are increased frequency of heart action and suddenly recurring attacks of palpitation and pain referred to the lower portion of the precordial region. The action of the heart is not only rapid, but at times is arhythmic; the first sound is short and sometimes sharp, resembling the second sound, and at other times it is deficient and hardly recognizable. In a sense every form of cardiac arhythmia might be described as irritable heart, but the "irritable heart" here referred to has peculiar features. Primarily, it is probably a functional disturbance of the vagus, but in the end degeneration of the cardiac muscle may take place. It should be treated in the first place by the careful avoidance of everything which tends to provoke cardiac excitement. Emotional outbreaks and abrupt unusual exertion should be especially avoided. Even after the affection has been long established, much good can be done by prolonged physical and mental rest. Irritable heart responds in some cases to cardiac depressants, and in others to cardiac

tonics. Probably these differences in response depend in part upon individual idiosyncrasies, and in part upon the degree in which the affection is structural or still simply functional. While any toxic agent may so affect the pneumogastric nerve as to cause an irregular or irritable heart, the effects of tobacco in this direction are so positive, and are of so much clinical importance, that it may be best to say a few words about the "tobacco heart." Chronic nicotine poisoning, as it is found in smokers, and only occasionally in tobacco workers, is not always well adapted to throw much light on this subject, for, whereas it is well known that the nicotine when brought into direct contact with the nerves paralyzes them rapidly, it is by no means common to find paralysis of the vagus in the course of nicotine intoxication. As a rule, it is true that the heart's action is increased, yet cases occur in which there is a slowing, so that we are led to think of a stimulation of the vagus such as happens after drinking cold water, when the pulse rate may be reduced to thirty or twenty beats. (Hirt.)

Nervous Palpitations.—Under the name of "nervous palpitations" several disorders, usually simple symptomatic affections, are described. The neurasthenic, hysterical, anemic, and debilitated, for example, under slight excitement or exertion, are very likely to suffer from annoying palpitations,—rapid, irregular beatings of the heart, which may be both objective and subjective. They can, in some instances at least, be regarded as functional disorders of the pneumogastric; or they are due to withdrawal of the normal cerebral control from the vagus centres; or they may be dependent upon an increased afflux of blood to the vagal centres as a result of functional and transient vasomotor paresis. When distress accompanies the palpitation, camphor or Hoffmann's anodyne may be given in full doses, and in some cases the persistent administration of small doses of tincture of aconite or of tincture of digitalis will be found useful. Any other treatment must be directed to the apparent cause, which is often gastric disturbance or emotional excitement.

DISTURBANCES OF THE DIGESTIVE ORGANS DUE TO DISEASE OF THE VAGUS.

As the pneumogastric nerve is distributed to a large portion of the gastrointestinal tract, to the liver, and probably to other organs concerned in the digestive processes, lesions and disorders of this nerve probably cause a considerable number of diseases not yet well studied. Paralysis of the esophagus may be due to a destructive lesion of one portion of the pneumogastric which supplies this structure. It is exceedingly rare as an isolated condition, but is somewhat frequently associated with palatal and pharyngeal paralysis. When the esophagus is paralyzed, the food which passes into it remains in its cervical portion or is regurgitated into the mouth. When it

remains in the esophagus it may by pressure cause dyspnea and other evidences of interference with laryngeal respiration. *Esophagismus*, or spasmodic stricture of the esophagus, may be caused by inflammatory or any other form of irritative disease of the pharynx or esophagus, or it may be due to a focal irritative lesion affecting the nerve supply of the esophagus. It is exceedingly rare as a result of central lesion. Hysterical spasmodic stricture of the esophagus is a somewhat common disorder. Globus hystericus is referred by some authorities to such spasm. Besides paralytic and spasmodic affections of the esophagus, gastralgia and nervous dyspepsia of various types belong in this category. True gastralgia is a painful affection of the stomach for which no adequate organic cause can be found. It is characterized by sudden and severe paroxysms of pain in the epigastric region, radiating in various directions, usually upward and backward. The pain is intermittent, or at least varies in intensity from time to time. Often it can be relieved by firm uniform pressure. Widespread vasomotor changes may accompany the paroxysms, the face becoming pale or even livid, and the extremities cold. The pulse is often small and feeble. The patient has a feeling of anguish, with great apprehension of impending dissolution, much like that which is experienced in severe attacks of angina pectoris. The attack sometimes terminates with vomiting. The diagnosis is to be made by the absence of evidences of organic disease of the gastrointestinal tract. Sometimes pain is relieved by the ingestion of food, in which respect the affection differs from other organic diseases, such as ulceration and inflammation of the stomach. It is more common in youth than in advanced years, and in females than in men; and it is often associated with anemia, chlorosis, or hysteria. Attacks of gastralgia bear some resemblance to the gastric crises of tabes which are among the most distressing tabetic episodes. Such crises were present in the case of Grabower, already referred to, in which the autopsy and microscopical examination showed, among other lesions, degeneration of the vagus nucleus. In the treatment of gastralgia the cause must first be sought and attacked. This may reside in the general condition of the patient, who may need to be treated for anemia, hysteria, or neurasthenia. Arsenic in small doses long continued is one of the most serviceable remedies for permanent effect. The diet should be carefully regulated, but this does not mean that the patient should be "dieted," in the sense that the total amount of food taken should be largely restricted. Often benefit is obtained by feeding the patient more than the usual number of times daily, seeing, however, that the food is carefully selected and is of the most digestible character. Blisters to the epigastrium sometimes prove serviceable, both in relieving special attacks and in preventing their early recurrence. For the relief of the paroxysms it is nearly always necessary to resort to hypodermatic

injections of morphine. By some, nervous dyspepsia is regarded as a pneumogastric neurosis, a disease due either to want of sensory innervation or to lesion or disturbance of the terminal filaments of the pneumogastric in the stomach. The term nervous dyspepsia as used in recent literature is chiefly due to Leube, and, accepting his views, any of the so-called gastric neuroses may be combined in a case of nervous dyspepsia. These gastric neuroses are disturbances of sensation, secretion, and motion. As described by Hirt, nervous dyspepsia, which is especially common in females and neurasthenic men, is characterized "by a loss of appetite, painful sensations in the region of the stomach, frequent vomiting, and still more frequent belching; besides these, the patients generally suffer from other nervous symptoms,—dull headache, vertigo, palpitation; they are easily tired, complain of a lump in their throat (globus hystericus), at times have a voracious appetite, and obstinate constipation is seldom absent. In rare cases periodical spells of vomiting have been noted (twenty to thirty in the twenty-four hours), accompanied by acute circumscribed swellings of the skin (angioneurotic œdema.)" The treatment should be particularly directed to improving the nutrition and the nervous tone of the patient. Good food, fresh air, sea bathing, hydrotherapy, and sufficient recreation are important measures. Arsenic is the most important internal remedy.

PNEUMOGASTRIC DISORDERS INVOLVING DIFFERENT VISCERA.

Lesions involving to a greater or less degree the entire trunk of the vagus or its centres, and toxic or other influences affecting them, may cause serious disturbances both in cardiac action and in respiration. Usually the most important cardiac affection has been tachycardia, and the most important respiratory disorder tachypnea with emphysema, often with catarrhal symptoms. What appears to be a true spasmodic asthma may be associated with tachycardia. A case of conjoint cardiac and pulmonary affection was found by Tuczek to be due to pressure exerted upon the trunk of the vagus by a rapidly swelling lymph gland. Van Noorden has observed a number of functional disturbances in hysterical females in the three separate areas supplied by the vagus, namely, (1) for the pharynx and larynx, hyperesthesia or anesthesia, and occasionally aphonia; (2) for the stomach, hyperesthesia, sometimes manifesting itself by pain and at other times by frequent vomiting; (3) for the heart, slowing and irregularity of its beat. He distinguishes four varieties of arhythmia as indicated by the pulse: (1) an intermission of a beat or a half beat in an otherwise normal pulse; (2) rapid and sudden change in frequency; (3) great irregularity in the pulsations, and (4) regular arhythmia, as double or triple pulse. In all Van Noorden's cases the stomach was the main cause of complaint.

DISEASES OF THE SPINAL ACCESSORY NERVE.

Varieties.—The spinal accessory nerve (the anatomy of which was discussed in the previous section) is a purely motor nerve, and hence its diseases can be classed under the two general heads of paralysis and spasm, according as the lesion affecting the nerve is destructive or irritative. In rare instances paralysis and spasm in the distribution of the accessorius may be combined in the same case, the destructive effects of the lesion in such a case being, of course, only partial. A spasm may be complete or incomplete; in other words, it may involve both the sternocleidomastoid and trapezius muscles, or either of these muscles may be affected separately. As the nuclei of those portions of the spinal accessory nerve which are related to the sternocleidomastoid and the trapezius muscles are more or less separated, and as the branches of this nerve distributed to these muscles distinctly part company for a considerable space before reaching their ultimate destination, and as the two muscles, while often and perhaps usually acting together, do sometimes have different physiological actions, it follows that limited destructive or irritative lesions may cause paralysis or spasm confined to either muscle, although in the majority of cases the affections of these muscles will be combined. Both paralysis and spasm of the accessorius are not infrequently combined with similar diseases affecting closely related cranial and spinal nerves, as the hypoglossal and vagus and the upper cervical nerves.

ACCESSORIUS PARALYSIS.

Symptomatology of Accessorius Paralysis.—*Isolated Paralysis of the Sternocleidomastoid Muscle.* Limited sternocleidomastoid paralysis may be either unilateral or bilateral. In paralysis limited to the sternocleidomastoid muscle of one side the patient's head is held obliquely, with the chin elevated and turned towards the paralyzed side. The ability to move the chin towards the nonparalyzed side is impaired; the sternocleidomastoid muscle, acting singly, draws the head towards the shoulder and clavicle of the same side, while the chin and face are carried towards the opposite side, away from the affected muscle. When the disease has lasted some time an actual flattening and loss of substance can sometimes be readily discerned. In the early stages of the affection little change is noted in the sternocleidomastoid muscle of the unaffected side, but when the disease has become chronic, contraction of this muscle occurs and the head is permanently carried in a twisted position, giving one of the paralytic forms of chronic wry neck. When both sternocleidomastoid muscles act together the face and head are bent forward towards the breast. When, therefore, bilateral paralysis of these muscles is present, if the head is kept quiet it may remain straight, but when

efforts are made to rotate it this can be done only with great difficulty, particularly when the chin is elevated. The prominences of the neck caused by these powerful muscles, usually so evident, are absent, so that the anterior lateral portions of the neck have a flattened appearance. The patient loses largely the power of making forward and backward nodding movements.

Isolated Paralysis of the Trapezius. The trapezius muscle has apparently a double nerve supply, only its upper portion being from the accessorius, while its lower part is supplied from the cervical cord. This is an important anatomical factor, and probably accounts for some of the peculiarities of trapezius paralysis and atrophy. In amyotrophic lateral sclerosis or chronic progressive muscular atrophy only the lower portion of the trapezius muscle may suffer, or at least the upper part does not degenerate until a very late period. Properly speaking, therefore, the nucleus of the spinal accessory, as this nucleus is generally recognized, is not attacked. With the exception, however, of pointing out the peculiarities of the nerve supply to the trapezius and their results, it will be most practical to speak of trapezius paralysis under the head of accessorius disease. The bulky trapezius muscle has several sets of fibres which join to produce movements in different directions. The upper fibres draw the shoulder upward and backward, the middle directly backward, and the lower backward and downward. As the upper fibres of the trapezius draw upward the outer end of the clavicle and the point of the shoulder, when these are paralyzed the clavicle will fall and project somewhat, making the supraclavicular space deeper and more prominent than natural, and at the same time the acromion process falls downward and forward and the body of the scapula will be pulled downward and forward by the action of the unaffected muscles. The same set of fibres acting from below flex the neck to the same side, extend the head, and turn the face to the opposite side, but unilateral paralysis of the muscle will not cause any special deformity, as the muscle of the other side and the muscles of the same side which are associated with the trapezius in action will tend to keep the head erect and in the middle line. Elevation of the arm above the horizontal line is extremely difficult, even when the paralysis is confined to the upper portion of the trapezius. The reason for this is that the deltoid loses to some extent its base of support or point of resistance. Close examination, however, shows some alteration in the shape of the neck, and some loss of power, especially on deep inspiration, in this act the head naturally tending backward as the lungs expand. The middle fibres draw the scapula inward towards the spine, producing at the same time a rotation of the scapula on the thorax, the result being an elevation of the point of the shoulder. If, therefore, these are paralyzed, the scapula inclines to fall outward. The lower fibres of the trapezius draw the scapula

downward and inward, at the same time rotating and raising the point of the shoulder. When, therefore, they are paralyzed, the result is that the scapula falls outward (Fig. 447). The rhomboid muscles being unopposed, they draw the lower end of the scapula inward and backward and at the same time somewhat upward. In bilateral paralysis of the trapezius muscle the head tends to fall forward and both shoulder blades to drop outward.

Fig. 447.

Posterior view of peripheral paralysis of trapezius. (Eichhorst.)

Complete Accessorius Paralysis (Combined Paralysis of the Sternocleidomastoid and Trapezius Muscles). Most frequently both the sternocleidomastoid and trapezius muscles are paralyzed together. The lesions affecting these nerves are usually such as to destroy the entire nucleus, or to compress or destroy the nerve before the branch to the trapezius parts company with that to the sternocleidomastoid. When both muscles are paralyzed together the symptoms described for each are present, but the symptomatology has some special features. In unilateral paralysis of both sternocleidomastoid and trapezius—unilateral spinal accessory paralysis—the head is rotated towards one side, the face and chin being turned upward towards the other, but at the same time it falls somewhat forward to the affected side, the clavicle and scapula assuming to a greater or less extent the positions already described as present in cases of isolated paralysis of each of the muscles. In the act of elevating the arm the patient throws his trunk towards the opposite side, and with the assistance of the action of the levator anguli scapulæ and of the rhomboid muscles he can succeed in elevating the shoulder and moving the scapula towards the vertebral column. When the combined paralysis is bilateral the head is so largely devoid of support that it

cannot be held erect. The paralysis of the sternocleidomastoid muscles allows it to fall backward and that of the trapezius muscles to fall forward. When the patient is sitting or standing, the head may tumble about in various directions. The patient is often observed placing his hand under his chin and pressing his head backward a little beyond the middle line. By carefully holding himself erect and maintaining this posture he can hold the head in this position, but a slight movement will cause it to fall forward on the chest. As soon as the head has passed to a certain point in its forward movement it drops suddenly to the breast with the movement which Lloyd has compared to the snapping shut of the blade of a penknife. Bilateral paralysis of these two great muscles from a focal lesion is extremely rare. It is comparatively common in cases of chronic muscular atrophy or amyotrophic lateral sclerosis.

Etiology and Pathogenesis.—One of the causes of peripheral spinal accessory paralysis is injury. This may be accidental or intentional, and of any character, as a blow, a fall, or a gunshot wound. According to Seeligmüller, watercarriers are particularly liable to suffer from accessorius paralysis, for the double reason that they are exposed to frequent wettings and that they carry their buckets suspended from their shoulders, and in this way the nerve trunk, the nerve endings in the muscles, or the muscles themselves are injured. (Hirt.) Accessorius paralysis is said to arise occasionally from injury of the nerve or its nucleus during difficult labor. Syphilis, rheumatism, diabetes, and infectious diseases are all causes of spinal accessory paralysis and spasm. The nuclei of the spinal accessory nerve may be attacked by inflammatory, gliomatous, or degenerative processes; the nerve roots at their origin may be implicated in a meningitis, in a cervical tumor, an exostosis, or vertebral caries, or in an aneurism or other lesion about the level of the foramen magnum. An injury, a growth, or an inflammation may involve the nerve anywhere in its peripheral distribution. The nerve endings in the nuclei may be affected by some toxic or infectious process. A lesion involving the nucleus of the nerve would produce paralysis and eventually atrophy, and if irritative would cause spasmodic phenomena. Degenerative and retrogressive lesions (gliosis) usually cause only paralysis and atrophy. Occasionally such nuclear lesion picks out the sternocleidomastoid or the trapezeus distribution alone. Lesions involving the nerve filaments or nerve trunk in its intravertebral course are usually associated with other symptoms of cervical disease, symptoms indicating involvement of the nerves of the cervical plexus, for instance, or the phrenic nerve. We have no data regarding accessorius paralysis due to cortical or subcortical lesion.

Diagnosis, Prognosis, and Treatment.—The diagnosis of accessorius paralysis, whether isolated to the sternocleidomastoid or the trapezius or affecting the entire distribution of the nerve, is made

by a critical study of the symptoms which indicate impairment or loss of physiological action of the muscles concerned. The positions of the head, of the clavicle, and of the scapula will be of particular assistance in making this diagnosis. Paralysis in the domain of the accessorius can usually be readily differentiated from spasm in the distribution of the same nerve. The only difficulty likely to arise would be in overlooking to a certain degree paralysis when both spasm and paralysis are associated in the same case. The prognosis will depend upon the character of the lesion. If the affection is due to nuclear degeneration it is, of course, unfavorable; but if dependent upon a peripheral lesion, neural or muscular, persistent treatment may prove successful. The treatment must be directed by a study of the cause of the paralysis. Nuclear cases can sometimes be stayed in their progress by the use of strychnine and by tonic and hygienic measures. Syphilitic and other focal lesions may be benefited by absorbent remedies. In peripheral lesions the use of counterirritants, of electricity, and of strychnine either internally or by hypodermatic injection, is most efficient.

SPASM IN THE MUSCLES SUPPLIED BY THE SPINAL ACCESSORY NERVE (SPASMODIC TORTICOLLIS).

Varieties of Spasmodic Torticollis.—Torticollis, or wry neck, may be due to a variety of causes. The most common form is the so-called rheumatic wry neck, in which the patient suffers from an inflammation, usually acute or subacute, involving the nerves, muscles, and perhaps other tissues of the neck. The spinal accessory nerve or the muscles supplied by it may, like other structures, be involved in an affection of this kind, but the rheumatic disorder is more properly considered in other connections. The forms of spasmodic torticollis which properly belong to the discussion of diseases of the spinal accessory nerve are those which are due to lesions or disturbances of this nerve, its centres, or its encephalic tracts. Several varieties of motor disorder properly classed as spinal accessory spasm can be recognized. The disease may be *acute* or *chronic*, *clonic* or *tonic*. The clonic form may be either unilateral or bilateral. Theoretically the tonic form may also affect the muscles of either one or both sides, but as a matter of clinical experience it is almost invariably unilateral and generally limited to the sternocleidomastoid. Partial or complete forms of spinal accessory spasm occur. In the former either the sternocleidomastoid alone, or the trapezius alone, or only a single portion (usually the upper portion) of the trapezius, may be affected, giving rise to *sternocleidomastoid spasm* or *trapezius spasm*. The clonic and tonic forms of the spasm are combined in the same case in one of the best known varieties of the disease.

Symptomatology.—In the unilateral tonic variety of accessorius

spasm the head is rotated so as to draw the occiput towards the shoulder of the side affected by the spasm, the chin being turned upward and towards the opposite side. Long continuance of this tonic spasm leads to a marked cervical spinal curvature. When the trapezius alone is affected by tonic spasm the head will be drawn backward and the point of the shoulder will be elevated. Attempts to bend the head forcibly forward will cause pain in the affected muscle. The symptomatology of the unilateral form of clonic spasm of the muscles supplied by the spinal accessory nerve will vary somewhat according to the severity of the spasm. In the severe type the face may be involuntarily twisted to the left and the chin thrown upward towards the side opposite to the spasm, while the occiput is drawn downward and towards the side of the spasm; sometimes the shoulder and arm take part in the spasm. Sometimes these violent spasms are rhythmical and repeated with greater or less rapidity. After the head is forcibly rotated to one side and backward as described, the muscles relax, it returns to the usual straightforward position, and a second spasm occurs, and so on throughout a long series. Early in the history of the case the intervals between the spasms may be comparatively long, but if the disease continues and increases in severity they become shorter and shorter. In one of my cases the spasms amounted to fifty and upward in a minute. In some cases the patients can arrest the spasm by a voluntary effort, but often this is impossible. The patients, however, in all except the most violent cases, after the disease has continued for some time seem to acquire a certain degree of tolerance. The spasms are usually less when the patient is sitting down, and sometimes can be controlled entirely when in the recumbent position; as sleep approaches they become less severe, and they usually disappear entirely during sleep. In Fig. 448 is shown a good example of true torticollis, recorded by Walton. The spasm of the sternocleidomastoid is plainly visible. A congenital form of spasmodic torticollis is always a tonic spasm. The condition is usually noticed soon after birth. Sometimes the affection is due to rupture of either or both of the muscles supplied by the spinal accessory, and in these cases

Fig. 448.

Spasmodic torticollis, showing prominence of the sternomastoid muscle. (Walton.)

it may be possible to observe by palpation the site of the rupture. If the condition is not remedied, permanent deformity is left, owing to the shortening of the muscles. An illustration of this variety of spasmodic torticollis is seen in Fig. 449. Spasm in the muscles supplied by the spinal accessory nerve may be associated with various other forms of motor spasm, especially those which affect the cranial motor nerves. Thus it is seen in combination with facial (seventh nerve) spasm, with masticatory spasm, with spasm of the tongue, and with spasm in the muscles supplied by the upper cervical nerves. Bilateral spasm of the accessory nerve is usually confined to the sternocleidomastoids. In this affection the head is jerked forward and downward, the chin being turned towards the sternum. If it is of the tonic variety, the head will be held in this position. If it is of the clonic variety, the head will be pulled downward by the spasm in the muscles, and thus relaxing will be carried to the erect position again by the normally acting muscles, when the spasmodic movement will be again repeated, and so on with a more or less frequent and continued series. Unless the lesion causing the spasm is destructive or degenerative, the electrical changes will not be present in the muscles affected, except, perhaps, in rare cases some hyperexcitability to the galvanic current. One of the varieties of head banging or head nodding (*eclampsia nutans, salaam convulsion*) is sometimes spoken of as bilateral clonic wry neck. It is met with almost exclusively in young children. It is a paroxysmal affection, the attacks of spasm lasting from a few seconds to a few minutes. It may recur, however, a number of times in the same day. Often during the attacks the child seems more or less bewildered, but consciousness is not lost.

FIG. 449.

The usual attitude in the so-called congenital cases of spasmodic torticollis. (Bradford and Brackett.)

Etiology and Pathogenesis.—When speaking of the etiology of accessorius paralysis, reference was made to focal lesions, which, according as they were destructive or irritative, might cause either paralysis or spasm. Accessorius spasm may be due to focal irritative lesions in a number of locations, as (1) in the spinal cord anywhere above the fifth or sixth cervical segment; (2) an intraspinal but extramedullary lesion, of membranes, bone, or nerves, in the upper portion of the vertebral canal; (3) in the postoblongata;

(4) in the main trunk of the accessory nerve as it passes downward to the sternocleidomastoid and trapezius muscles; (5) in any of the nerves which anastomose with the spinal accessory and are closely connected with it in the spinal cord; (6) in the cortical centres which preside over lateral deviation of the head. In one of my cases it seemed to me that the spasm was reflex, due to injury and subsequent inflammation of either the major occipital or the minor occipital nerve. The spasm had come on in this case after an injury to the back of the head. Both the occipitalis major and the occipitalis minor have close connections with the spinal accessory nerve, and are also topographically in close relation, the former with the trapezius and the latter with the sternocleidomastoid muscle. Both the sternocleidomastoid and trapezius muscles have their main nerve supply from the second and third cervical segments. The occipitalis major nerve is the internal branch of the posterior division of the second cervical nerve; and the occipitalis minor also arises from the second cervical nerve. The occipitalis major nerve pierces both the complexus and trapezius muscles near their cranial attachments. The occipitalis minor in its course curves around the posterior border of the sternomastoid and then ascends along this muscle for some distance. Both of these nerves are, therefore, closely connected both inside and outside of the spinal canal with the nerve supply to the sternocleidomastoid and trapezius, and, in addition to having some direct anatomical relations with the muscles themselves, possibly supply them with some filaments. The motor nerve supply from the occipital nerves is also such as to show how their irritation might give rise directly or by anastomosis to varying spasmodic phenomena. The posterior division of the second cervical nerve, from which the major occipital originates, supplies the inferior oblique muscle; and the external branch of the major occipital joined by the external branch of the posterior division of the third cervical nerve supplies the complexus, splenius, and trachelomastoid muscles. Féré has reported a case of spasm of the neck associated with optical illusions which he believed was due to a cortical irritation, both motor and sensory. Another case has been recorded in which the spasm was associated with auditory disturbances.

Diagnosis.—In the diagnosis of accessorius spasm it is sometimes important to distinguish it from spasm of adjacent or related muscles. It might be a question, for instance, whether the spasm was of the sternocleidomastoid of one side or of the splenius capitis of the other. In a case which came to my service at the Philadelphia Polyclinic, the chin did not turn upward and to the left, as is the case in spasm of the sternocleidomastoid, but rather outward. In spasm of the splenius capitis the chin is directed rather towards the corresponding side. The diagnostic symptoms of spasm of the

splenius capitis as given by Erb are as follows: "In this affection the head is drawn backward towards the affected side, the chin is somewhat depressed and directed towards the corresponding shoulder, and at the spot where the splenius appears beneath the anterior border of the trapezius a hard roll can be felt. (The diagnosis of this form of spasm of the trapezius is founded on the fact that in this last the head is rotated towards the opposite side. In spasm of the sternocleidomastoid the chin is raised and rotated towards the opposite side, while the mastoid process is drawn forward and downward.) This spasm is for the most part of a tonic character (with remissions and occasional spasmodic contractions, or appears in the form of a permanent contracture." Some forms of spasmodic horizontal rotation of the head need to be differentiated from accessorius spasm. In spasm of the obliquus capitis inferior, for instance, the head is rotated either intermittently or persistently around its vertical axis without any elevation of the chin or depression of the mastoid process. The patient suffering from this form of spasm is sometimes obliged to hold the head in its natural position in order to look at a fixed object or to speak; or he may be observed to correct the peculiar position of the head with the hand when walking. (Erb.) When the head is thus rotated intermittently the affection is spoken of as *tic rotatoire.* It may be an hysterical or a hysteroneurasthenic phenomenon. One of my patients when overexcited or depressed was liable to violent attacks of this form of rotatory spasm. Her head would be rotated horizontally with great rapidity and force, the spasm sometimes being simply this form of motion, and at other times accompanied by a rapid spasmodic crossing of the arms, and occasionally by movements of the legs and body. I have known this distressing rotatory spasm to be kept up for three hours. During the attack the patient remained clear-headed, and, considering the violence of the movement, comparatively placid. The spasm could be stopped or abated by forcibly holding the head; at other times this procedure would lead to greater violence of action in the trunk and limbs. The patient would sometimes converse during the continuance of the spasm, the rotation diminishing a little while she was talking. Strong mental impressions were very effective both in inducing and in stopping the spasm. When it had once occurred it had a tendency to recur about the same time the following day; and this could often be prevented by giving a nerve tonic or other medicine, with a positive assertion that it would prevent the attack.

Prognosis.—The measures usually most serviceable in the treatment of spasm in the distribution of the spinal accessory will be given, but it is necessary to say that it is one of the most intractable of all spasmodic diseases. Absolute cure is brought about in very few cases, except those which are of hysterical origin or are manifestly due to a rheumatic neuritis.

Treatment.—If spasm in the muscles supplied by the accessorius is due to an irritative lesion in the course of the nerve, the treatment should be directed to the removal of this lesion or to rendering it as harmless as possible. If dependent upon a neuritis, meningitis, or a tumor, mercury and the iodides may prove of great benefit, and this even in cases in which no syphilitic lesion is present, although the results to be obtained from this treatment are, of course, more marked when syphilis is the cause. The salicylates are of special value when spasmodic torticollis is due to a true neuritis. Among other measures of proved value in these cases is the use of derivatives and counterirritants. Blisters, mustard applications, thapsia or capsicum plasters, and, above all, the use of the actual cautery, have proved highly efficient in recorded cases. The application should be made to the nape of the neck or over the course of the nerves. The actual cautery in several cases has proved in my hands to be one of the best methods of direct treatment. The only objection to its use is the dread in which it is sometimes held by patients or their friends, but this can generally be overcome. I prefer the method of superficial application. A small blunt applicator should be used, and this heated to whiteness is passed rapidly over the skin for the distance of from half an inch to an inch. Treatment can be repeated every day or every other day for the first week or two, making the application in different positions from the middle of the nape of the neck towards the side on which the spasm occurs. Some of the older works advocate the use of deep applications to each side of the spinal column in the cervical region, afterwards maintaining suppuration for some weeks, but this is a more uncomfortable method than that of making superficial burns and frequently repeating them, and the reported results are not better than those which have been obtained in the less annoying mode of treatment. In one of my cases of very severe type this treatment succeeded after other measures had been tried for many months. Among remedies employed for the direct purpose of controlling the spasm are preparations of atropine, hyoscyamus, conium, gelsemium, morphine, cannabis indica, and Calabar bean. On the whole, I have obtained the best results from the use of preparations of gelsemium and coniine pushed to their constitutional effects. I have used both conium hydrobromate and hyoscine hydrobromate with temporary benefit, the former in doses of from one thirtieth to one fifteenth of a grain, and the latter in amounts varying from one hundred and fiftieth to one hundredth of a grain. Hypodermatic injections both of atropine and of morphine have had their advocates, and the administration of these remedies will usually temporarily abate the spasm. The patient is always in danger of establishing a drug habit in an affection so recalcitrant to all treatment. Other remedies which have been used with transient and varying benefit are the bromides, chloral, and chloralamid, in

comparatively large doses, and inhalations of amyl nitrite. Salts of gold, silver, zinc, copper, iron, and arsenic, with nourishing diet and hygienic measures calculated to improve the general health, should be employed in conjunction with the remedies especially directed to the relief of the spasm. All sources of reflex irritation should, if possible, be removed. Now and then a striking success will be scored by the treatment of diseased teeth or gums, or of an affection of the ear or of some other part of the face and head. The electrical treatment of this form of spasm has been a matter of much interest, and many reports have been made upon the results. Faradism, galvanism, and franklinism have all been used. The best method of electrical treatment is the steady application of the anode of a galvanic current over the accessory nerve, the cathode being placed at some indifferent spot. Poore's method of treating writer's cramp with the galvanic current and rhythmical exercise of the affected muscles is well worthy of trial. Apparatus to hold the head in proper position may be of service if conjoined with other treatment, such as faradization of the antagonistic muscles. Nerve stretching, myotomy, neurotomy, and neurectomy are operations which may be performed for the relief of spasmodic torticollis. Both neurotomy and neurectomy have been employed with success; but in a greater number of instances they have failed. The fact that the spinal accessory is not the only nerve supply to the muscles chiefly accounts for the failure of the operation. One surgical operation which has met with some success in the hands of Keen and others is resection of the posterior branches of the upper cervical nerves. The operation, according to Risien Russell, is the only measure which offers a reasonable prospect of permanent relief in severe cases. Russell found considerably more individual variation with regard to the nerve root supply of the neck muscles than of those of the limbs. Where the lateral inclination of the head, by which the shoulder and side of the head and face are approximated, exists, the first and second cervical nerve roots are those to which attention should be directed, while in those instances in which the chief movement of the head is one in which the occiput is drawn backward, so that the face looks more or less upward, the third and fourth cervical nerve roots should engage attention. Either primarily or secondarily other muscles besides those supplied by this nerve become involved in the spasm. Among the muscles supplied by the upper three cervical nerves are, for instance, the inferior oblique, the rectus capitis posticus major, the splenius, and the three posterior rotators. Good authorities have argued that not a few of the cases are of cortical origin and should be treated by trephining and the removal of lesions supposed to be present in the cortex or subcortex, or by excision of the unstable cortex; but the facts are not sufficient to encourage the endorsement of this treatment.

DISEASES OF THE HYPOGLOSSAL NERVE.

The Hypoglossal Nucleus.—The hypoglossal nerve arises from a large mass of cinerea, sometimes called the chief nucleus, which is in part situated on the ventral side of the most encephalic portion of the central spinal canal and in part in the gray matter of the floor of the fourth ventricle. The spinal portion of this nucleus is a portion of the ventral horn which remains attached to the central gray matter after the decussation of the pyramidal fibres. (Obersteiner, Koelliker.) Cephalad it reaches to the acoustic striæ. It contains very large multipolar cells like those found in the ventral horn of the cord. It is evident from its microscopic structure that the hypoglossal nerve is almost entirely, if not purely, motor in its functions. The hypoglossal nucleus is solely a nucleus of origin. The axis cylinders of its large cells form efferent fibres which pass into the nerve of the corresponding side. According to Obersteiner, some fibres arise in the nucleus of the opposite side; but this has been denied by other investigators. Certain afferent hypoglossal fibres are derived from the pyramids, forming mainly what are known as the *fibræ propriæ*. Other efferent fibres come from the reticular formation, and possibly from the fillet. (Turner.) According to Turner, besides the above connections and relations of the hypoglossal nucleus, fibres enter the nucleus from the dorsal longitudinal bundle, and commissural fibres exist between the two nuclei. According to Ramón y Cajal, a decussation of the axis cylinders of the cells of the hypoglossal nucleus does not occur in the newborn mouse. Turner believes that these nuclei do not give origin to any hypoglossal fibres. In the mouse and rabbit the twelfth nerve arises only in the chief nucleus. He agrees with Koelliker in greatly doubting the connection of the twelfth with Roller's nucleus or Duval's accessory nucleus.* The twelfth centre in mice receives sensory fibres from the cells in the terminal nucleus of the ninth and tenth nerves, fibres from the substantia gelatinosa, accompanying the spinal root of the fifth nerve, and fibres from the common central tract of the fifth, ninth, and tenth nerves. Ramón y Cajal has not been able to observe direct collaterals passing from the roots of the fifth, tenth, and ninth to the twelfth centres, the connections seeming to be by the axis cylinders from cells in the terminal nuclei of these nerves.

Peripheral Course of the Hypoglossal Nerve.—The axis cylinder processes of the ganglion cells of the hypoglossal nucleus just

* What is termed the nucleus of Roller is a round but not distinctly circumscribed clump of small nerve cells which lies close up against the ventral side of the large-celled nucleus. Duval's accessory hypoglossal nucleus is a collection of large multipolar cells placed ventrolaterally to the chief nucleus.

described pass obliquely forward and outward between the latero-ventral regions of the postoblongata, to appear on its upper surface as a series of a dozen or more fine roots, which emerge in the groove between the anterior pyramid and the olive. These filaments commonly unite into two separate bundles, which pierce the dura opposite the anterior condyloid foramen, becoming a single trunk to pass outside of the skull through this foramen. In the first part of its extracranial course it is very deeply situated to the inner side of the

FIG. 450.

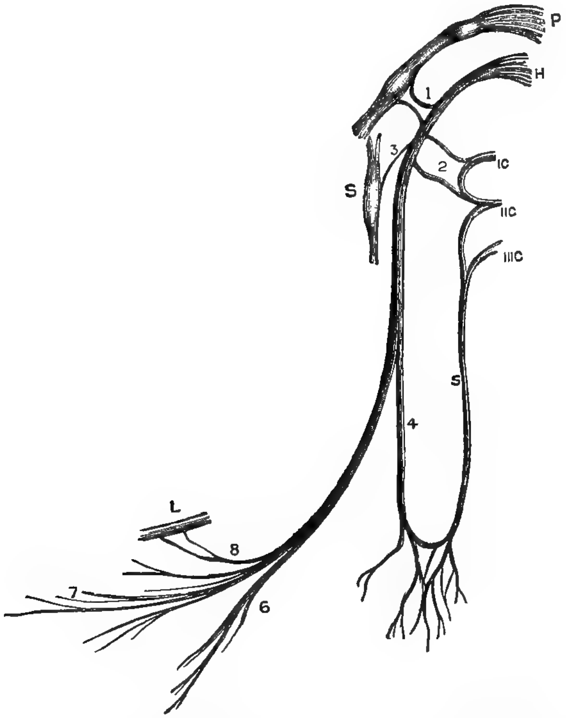


Diagram of the hypoglossal nerve, its connections and branches: H, hypoglossal nerve; P, pneumogastric nerve; S, superior cervical ganglion of the sympathetic; L, lingual nerve; IC, IIC, IIIC, the three upper cervical nerves; 1, communicating branches from hypoglossal to ganglion of the trunk of the vagus; 2, connecting filaments with the loop of first and second cervical nerves; 3, branch to the sympathetic; 4, descendens noni; 5, branch from second and third cervical nerves (communicantes noni); 6, branch to thyrohyoid; 7, terminal muscular branches; 8, communicating branch to the lingual branch of the fifth. (Ross.)

pneumogastric. It winds around the lower ganglion (ganglion of the trunk) of the pneumogastric, and descends, gradually inclining forward, between the internal carotid artery and the jugular vein, to the lower border of the digastric muscle. Here it curves and passes forward above the hyoid bone. Towards the end of its peripheral course it rests on the hyoglossus muscle and is accompanied by the ranine vein of the tongue. At the anterior border of the hyoglossus

it communicates with the lingual branch of the fifth. In the anterior condyloid foramen it gives off a few small branches which are distributed in the dura around the foramen magnum and to the occipital diploe. These fibres, usually classed as hypoglossal twigs by some authorities, are regarded by others as derived from the lingual, and by still others as fibres from the pneumogastric, the first cervical, or the sympathetic. Its communicating branches are with the superior cervical ganglion of the sympathetic, with the loop between the first and second cervical nerves, with the ganglion of the trunk of the vagus, with a small lingual branch of the vagus, and with the lingual branch of the fifth. It has three distinct sets of branches of distribution: (1) branches to the vessels (vascular branches); (2) descending cervical branches; and (3) muscular branches. The vascular branches pass to the mesal aspect of the jugular veins, where they are sometimes joined by communicating filaments from the cervical sympathetic. The cervical branches of the hypoglossus (*descendens hypoglossi*) run downward on or within the carotid sheath, and are joined by communicating branches from the second or third cervical nerve, which form with them a loop (*ansa hypoglossi* or *ansa cervicales*). A small branch passes to the omohyoid before it supplies this loop. Branches are given off from the ansa to the sternohyoid, sternothyroid, and posterior belly of the omohyoid. Twigs from the first two nerves pass in the muscles behind the manubrium sterni and in rare cases communicate with the phrenic within the thorax. (Morris's Anatomy.) A branch leaves the hypoglossal trunk as it approaches the hyoid bone to pass to the thyrohyoid muscle. This is composed in part of fibres from the upper cervical nerves. The nerve also gives off branches to the styloglossus, hyoglossus, and genioglossus. Terminal branches supply the genioglossus and the intrinsic muscles of the tongue. Both anatomically and physiologically it is somewhat difficult to discriminate between the branches of distribution which are derived from the hypoglossus nucleus and those which come from the nuclei of the upper cervical nerves. Quain thus summarizes the above facts: "The hypoglossal nerve proper supplies only the muscles of the tongue, with the exception of the palatoglossus and the pharyngeoglossus. Fibres derived from the first three cervical nerves, which are associated with the hypoglossal for a part of their course, are distributed to the infrahyoid muscles and the geniohyoid. Others of uncertain origin pass to the skull and dura mater and to the internal jugular vein. The hypoglossal forms connections with the pneumogastric, lingual, upper three cervical nerves, and sympathetic." The hypoglossal nerve is the chief motor nerve to the tongue, and also to the muscles which elevate and depress the hyoid bone. A dorsal root for the hypoglossal nerve has been found in some lower animals,—this nerve in them, therefore, more closely resembling a true spinal nerve.

HYPOGLOSSAL PARALYSIS.

Paralysis of the hypoglossus (*glossoplegia*) may be either unilateral or bilateral, but it is much more frequently the former. In unilateral hypoglossal paralysis the tongue at rest presents no features of importance. On protrusion it deviates towards the paralyzed side (Fig. 451), the reason for this being that the movements of protrusion are of a pushing or propelling character, and hence the tongue deviates to one side by the unopposed action of the muscles of the healthy side. The movements of the tongue within the mouth are also interfered with, the patient in consequence having difficulty in mastication and deglutition. The food is not carried backward into the pharynx with facility, although this may not be very noticeable in cases of one-sided paralysis. When the paralysis is bilateral all the movements of the tongue are affected, and if the paralysis is complete the tongue lies motionless within the buccal cavity. As this form of paralysis is almost invariably the result of nuclear degeneration, atrophy and fibrillary twitchings are usually present in addition to the immobility (see Lingual Atrophy). When the patient is lying on his back the tongue may fall backward into the throat and partially close the glottis, thus interfering with respiration and leading to partial asphyxia and stertorous breathing. In incomplete bilateral paralysis of the tongue the extraordinarily intricate and complicated movements of the tongue, performed with such facility in health, are more or less interfered with according to the extent of the paralysis. Mastication and deglutition are of course much more seriously interfered with in bilateral than in unilateral paralysis. Food, drink, and saliva may collect in the cavity of the mouth and throat, annoying the patient and even at times threatening his life because of the interference with respiration. Speech is affected in both unilateral and bilateral hypoglossal paralysis. In bilateral cases the loss of speech may be complete. Careful studies have been made in the degree of impairment of articulation at different stages in progressive hypoglossal paralysis. "Articulation becomes indistinct, difficulty being first experienced in singing and in pronouncing

FIG. 451.

Unilateral paralysis of the tongue (probably due to degeneration of the hypoglossal nucleus of one side) in a case of beginning bulbar paralysis.

the letters s, sh, l, e, i, and, at a later period, k, g, r, etc., while if the paralysis be bilateral, complete, and associated with atrophy, vocal speech becomes quite inarticulate and unintelligible, this condition being named *anartheca.*" (Ross.) It is difficult to separate the etiology and pathogenesis of paralysis and spasm of the hypoglossus. The nerve at or near its origin may be involved in various pathological processes, such as meningitis, tumor, aneurism, or caries. In rare instances an abscess or a hemorrhage from one of the vertebral arteries has involved the nerve trunk. Occasionally the nerve is the seat of a neuritis of rheumatic, syphilitic, or unknown origin, but it is less frequently affected in this way than most of the other cranial nerves. Habershon has reported a case of cancer of the cervical vertebra with paralysis of the right hypoglossal nerve. Autopsy revealed a cancerous mass involving the basilar process. The nucleus of the hypoglossus is not infrequently the site of degenerative changes. The result of this is both paralysis and atrophy, the paralysis being proportionate to the degree of loss of substance in the tongue; but the effects of such degenerative disease will be more fully considered under lingual atrophy and bulbar paralysis.

HYPOGLOSSAL SPASM.

Hypoglossal spasm, known also as *lingual spasm* and *aphthongia,* is a comparatively rare affection. It is especially rare as an independent disorder, but it sometimes forms a part of the symptomatology of such more general affections as epilepsy, chorea, and hysteria. Trigeminal and hypoglossal spasm are not infrequently associated in the same case. Hirt reports a case in which the muscles of mastication took part in the affection in such a way that before the actual hypoglossal spasm occurred the lower jaw was for a half or a whole minute spasmodically jerked to and fro and up and down. "After these movements had ceased the mouth remained half open, and the turning and rolling movements of the tongue commenced and lasted for about one minute. These attacks recurred ten to twenty times a day; they came on for the first time three days after an epileptic fit." (Hirt.) Hypoglossal spasm may form a part of the syndrome of bilateral athetosis in one of the forms of infantile diplegia already described. Choreiform and tremulous movements of the tongue, which belong pathologically to the same general category as lingual spasm, are sometimes symptoms of progressive bulbar paralysis and of disseminated sclerosis. The symptoms of hypoglossal spasm vary as the affection is clonic or tonic or a combination of both. Muscles innervated by the nerve in one of the described forms of the disease are thrown into a state of clonic or tonic convulsions when the patient attempts to speak, the affection being more or less similar to writer's cramp. The spasm may implicate other groups of muscles than those supplied by the hypoglossus. In some cases the tongue becomes fast-

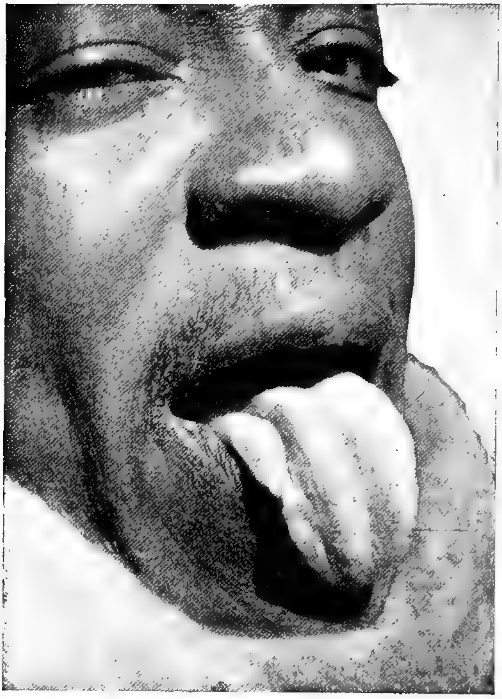
ened to the hard palate by a tonic spasm whenever the patient tries to speak, but in other cases it is attacked with clonic spasms, and the sternohyoid, thyrohyoid, and sternothyroid may likewise be implicated in the convulsion. (Ross.) Hypoglossal spasm, like hypoglossal paralysis, may be due to focal lesion anywhere from the nucleus to the peripheral distribution of the nerve. A few cases have been recorded in which the seat of the disease was apparently in the cerebral cortex about the lower extremity of the central fissure. The diagnosis is readily made, the tongue being the chief organ supplied by this nerve and therefore the one most affected, although the spasms may, as has been stated, attack the sternohyoid, thyrohyoid, and sternothyroid muscles. It would seem impossible that any other affection should be mistaken for this. The only difficulty would be in distinguishing hysterical from organic cases, and this would have to be done by a search for hysterical stigmata and a careful consideration of the probability of the presence of a focal lesion. Its treatment is that of local spasm elsewhere.

LINGUAL ATROPHY.

Mainly as the result of degeneration of the hypoglossal nucleus the tongue may be atrophied in whole or in part, although in rare cases, as just stated, this atrophy may accompany paralysis from peripheral lesions. The most distinctive forms of lingual atrophy are those which are in association with such progressive degenerative and retrogressive diseases as amyotrophic lateral sclerosis, chronic muscular atrophy, progressive bulbar paralysis, and syringomyelia. In the majority of these cases the nuclear degeneration, and consequently the atrophy of the tongue, are bilateral, and attain their maximum only after the disease has endured for several years. The characteristic features of lingual atrophy are wasting of the muscles of the tongue, with fibrillary tremors, and in some instances trophic phenomena, such as ulceration. Hemilingual atrophy as an isolated affection is extremely rare. Little needs to be said about its symptomatology more than has been said under hypoglossal paralysis. The tongue on one side is wasted, wrinkled, tremulous, and wanting in power. Lingual movements, speech, mastication, and deglutition are all more or less impaired. Among the most frequent predisposing causes of hemilingual atrophy is syphilis, although it is probable that it may arise from the influence of any toxic agent. Among the pathological causes to which it has been ascribed are traumatism, disease of the vertebral artery, new growths in the oblongata, and softening from an embolus or a thrombus in the region of the hypoglossal nucleus. While, as has been stated, hemilingual atrophy is usually part of the syndrome of a progressive degenerative disease, the degeneration in rare cases attacks only the hypoglossal nucleus. Westphal demonstrated an interesting specimen

from a patient who had had external ophthalmoplegia and complete paralysis of both eyeballs. He had also a bulbous appearance of the tongue, and an atrophy of the anterolateral portion of its left side. The specimen showed the right hypoglossal muscles to be well developed, while the others were only fragments. On the right side the root was distinctly seen running to the nucleus, but nothing could be seen on the left, except with a very high power, and then only a few nerve fibres. He also found a gray degeneration through the whole length of the cord. Fig. 452 is an illustration of hemilingual atrophy in a well marked case of syringomyelia. The diagnosis of hemiatrophy of the tongue can, as a rule, be readily made by a careful examination of the organ. The tongue may be congenitally smaller on one side than on the other. Lingual hemihypertrophy is exceedingly rare, and its presence might at first lead to the idea that the unaffected side of the tongue was atrophied. Fibrillary twitching is in favor of nuclear disease, but is not absolutely pathognomonic, as twitching has been recorded in peripheral disease. The localization of the lesion causing the lingual hemiatrophy, as well as the diagnosis of its nature, may be largely assisted by a study of the accompanying symptoms. When hemiplegia of the side of the body opposite to the atrophied tongue is present, the lesion is probably gross, and one of such size and character as to involve the pyramidal tracts before their decussation. Sensory and other symptoms are likely to be present in such cases. When scattered but considerable portions of the musculature of the body are affected conjointly with atrophy of the tongue, the lesion is probably one of a disseminated or diffused degeneration. The prognosis is unfavorable, and no treatment is of avail except in peripheral cases, in which absorbents and electricity may be of some service.

Fig. 452.



Unilateral atrophy of the tongue in a case of syringomyelia.

GROSS LESIONS OF THE POSTOBLONGATA AND OBLONGATA-SPINAL TRANSITION.

General Remarks on the Diagnosis of Lesions of the Postoblongata.—The method of diagnosticating small gross lesions in the pons and preoblongata was discussed in the last section of Chapter IX., where was given a series of topographical schemes and semidiagrammatic illustrations of the most important structures of these regions. Having considered the cranial nerves which take their origin from the postoblongata, it will be well, in pursuance of the same plan, to discuss briefly the localization of similar lesions in the postoblongata and oblongata-spinal transition. Points of diagnosis to which attention cannot be easily and readily directed when considering the separate nerves and neural cell nests can thus be elucidated. That such a discussion is not without practical value has been illustrated by cases which have fallen under my personal care, and by not a few which have been recorded in neurological literature. Operations for the removal of encephalic growths have been performed on regions of the cranial vault when autopsy has revealed tumors so situated as to press upon or invade the postoblongata; lesions of the Gasserian ganglion or of the trunks of the sensory subdivisions of the trigeminus have been diagnosticated when the real seat of the disease was in the descending (spinal) root of the fifth nerve and in the postoblongatal portion of its course; so serious a diagnosis as that of hydrophobia has been made when the real source of the so-called hydrophobic symptoms was an isolated lesion of the region of the oblongata-spinal transition; and the diagnoses of intracerebral hemorrhage and of acute bulbar paralysis have been made in cases of hemorrhage from the vertebral artery. As said by Krauss, to whom we are indebted for some valuable work on this region, the study of the transition from the spinal cord to the brain is perhaps the most difficult task in the anatomy and histology of the nervous system, requiring time and care to trace the origin and direction of the nerve bundles, the appearance of new masses of gray matter, and the coalescence of different tracts. The symptoms of gross lesions affecting this region are often as confusing as the structures are complicated; and yet it is not impossible to lay down rules which will assist in approximately fixing lesions in any one of five or six positions in the space which lies between the furrow separating the pons and postoblongata and the most proximal roots of the first pair of cervical nerves, just above which is the great pyramidal (motor) decussation. (Fig. 453.) In order to do this it is necessary to have at command a knowledge not only of the superficial anatomy, but also of the structures shown by transections made in at least four or five planes of this area. Descriptions of the external features of the ventral and dorsal aspects of the oblongata, of the floor of the fourth

ventricle, of the region of transition from the spinal cord to the oblongata, of the interior structure of the latter, and of the deep origins and roots of the cranial nerves, have been given in Chapter I. (pages 73–86); and it will, therefore, be necessary to recall here, with the assistance of some semidiagrammatic illustrations, only the most important of the structures there described, indicating at the same time the parts which they play in symptomatology.

FIG. 453.

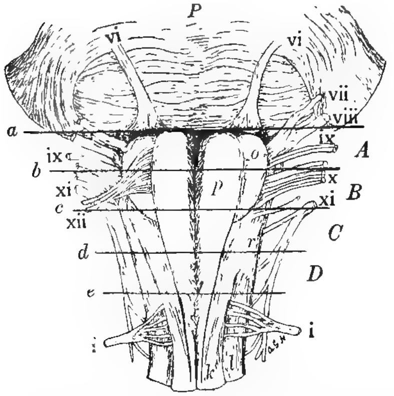


FIG. 454.

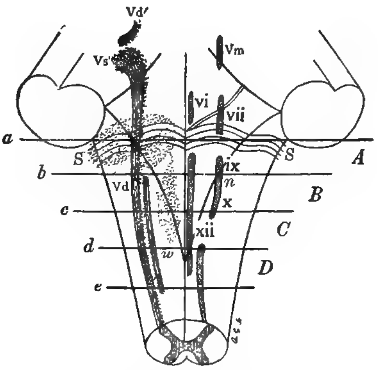


FIG. 453.—Subdivision of the ventral aspect of the postoblongata into fourths. The fourths are separated by the letters *a*, *b*, *c*, *d*, *e*. The diagrams of transections of the postoblongata which follow (Figs. 455, 456, 457, 458, 459) are made through about the middle of the sections *A*, *B*, *C*, and *D*. *P*, pons; *o*, olive; *p*, pyramid; *r*, restis; *i*, roots of the first cervical nerve; *l*, lateral column; *k*, anterior column. The Roman numerals indicate the cranial nerves.

FIG. 454.—Subdivisions of the dorsal aspect of the postoblongata: *n*, nucleus ambiguus; Vs, sensory nucleus of the fifth nerve; Vd′, descending (mesencephalic) root of the fifth; Vd, descending spinal root of the fifth; Vm, motor nucleus of the fifth; w, descending glossopharyngeal root. The Roman numerals indicate the nuclei and nerve roots.

Topographical Subdivisions of the Postoblongata.—In Figs. 453 and 454 the postoblongata is represented as subdivided into fourths by the lines *a*, *b*, *c*, *d*, and *e*, these fourths being designated by *A*, *B*, *C*, *D*, and *E*. The uppermost line, *a*, passes through the furrow which separates the pons from the postoblongata, and the lower through the motor decussation. The legends accompanying these diagrams sufficiently describe them. A series of five transections is also given (Figs. 455, 456, 457, 458, and 459). The first three of these illustrations represent transections of the postoblongata in its upper, second, and third fourths. A study of these diagrams shows that it would not be impossible to diagnosticate ventral or ventrolateral and dorsal lesions limited to any one or to any two or more of these subdivisions. An accurate limitation of a lesion to one of these subdivisions is not likely, but is not impossible, as, for instance, in the case of aneurism or small growths. The aneurism recorded by Daland (page 961) was of this character.

Effects of Limited Lesions of the Postoblongata.—Let us

briefly review the structures which would be implicated in limited areas of different portions of this complicated region, and the symptoms which would result from lesions more or less strictly confined to

FIG. 455.

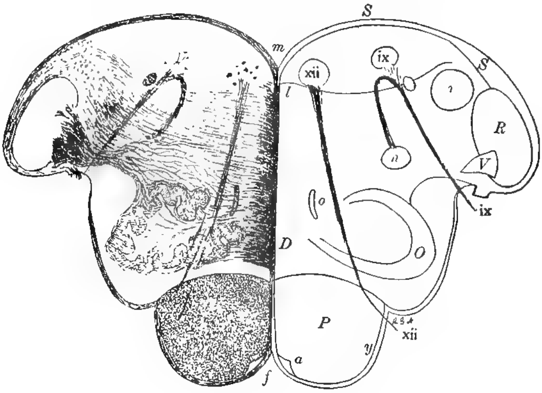


FIG. 456.

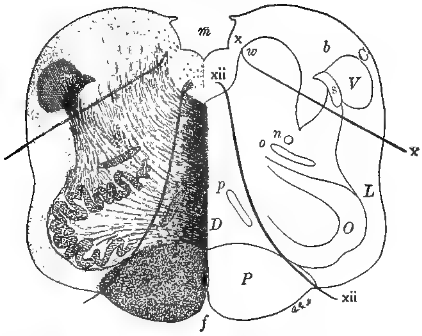


FIG. 457.

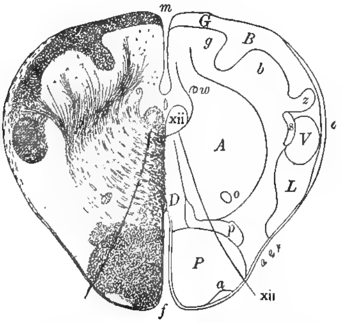


FIG. 455.—Transection of the postoblongata in its upper fourth (through *A* in Figs. 453, 454): *SS*, acoustic stria; *r*, descending root of eighth nerve; *R*, postpeduncle (restis); *V*, descending (spinal) root of the fifth at about the level of the fibres of the trapezoid body; *O*, olive; *P*, pyramid; *a*, arciform nucleus; *y*, external arciform fibres; *D*, lemniscus; *o*, accessory external olive; *n*, nucleus ambiguus; *m*, fourth ventricle; *f*, anterior median fissure; *l*, dorsal longitudinal bundle. In all the sections the Roman numerals indicate the nuclei and root fibres of the different nerves.

FIG. 456.—Transection of the postoblongata in its second fourth (through *B* in Figs. 453 and 454): *w*, descending glossopharyngeal root or vagoglossopharyngeal root; *b*, remains of the nucleus of the column of Burdach; *s*, substantia gelatinosa; *L*, remains of lateral column; *p*, nucleus of the pyramid (internal accessory olive, anterior accessory olive). (The other letters indicate the same structures as in Fig. 455.)

FIG. 457.—Transection of the postoblongata in its third fourth (through *C* in Figs. 453 and 454): *g*, nucleus of the column of Goll; *G*, column of Goll; *b*, nucleus of the column of Burdach; *B*, column of Burdach; *z*, accessory nucleus of the column of Burdach; *A*, internal arciform fibres; *D*, lemniscus; *c*, fibres of direct cerebral tract passing to restis. (The other letters indicate the same structures as on Figs. 455 and 456.)

them. In doing this, attention will be directed only to ventral or ventrolateral and dorsal subdivisions. Lesions restricted to the area intermediate between the ventral and dorsal thirds of the postob-

longata are conceivable, but must be of exceedingly rare occurrence. It might chance that a hemorrhage, or a focus of softening caused by an embolus or a thrombus, would be limited to this central portion of the interior of the postoblongata. It would cause symptoms which would chiefly show involvement of the lemniscus and trapezoid body, and perhaps of a portion of the olive and the nucleus ambiguus. A lesion in the ventral or ventrolateral portion of the upper fourth of the postoblongata (*A*, Fig. 454, and transection shown in Fig. 455) would involve the pyramid, inferior olive, arciform nucleus, external arciform fibres, and the root bundles of the twelfth, the ninth, and probably the eighth nerve. The syndrome would, therefore, chiefly be contralateral paralysis of the limbs, and clinical phenomena indicating implication on the same side of the nerves enumerated (see sections on the hypoglossal, glossopharyngeal, cochlear, and vestibular nerves). Lesions situated in the dorsal portion of the upper fourth (Fig. 455) would probably involve, in addition to the nuclei and root fibres of the glossopharyngeal and twelfth nerves, the dorsal longitudinal bundle, the postpeduncle, and the descending root of the trigeminus. The symptoms would therefore be sensory phenomena in the opposite half of the body, in the distribution of the fifth on the same side, cerebellar manifestations probably such as forced movements, and phenomena referable to the eighth, ninth, and twelfth nerves,—affections of hearing and taste, and paralytic or spasmodic disorders of the throat and tongue. In the second fourth of the postoblongata (*B*, Fig. 454, and transection shown in Fig. 456) ventrolateral lesions involve the pyramid, olive, fillet, remains of the lateral columns, and the root bundles of the twelfth nerve. The symptoms of a unilateral lesion should therefore be chiefly motor paralysis in the extremities of the opposite side, with evidences of hypoglossal disease. Dorsal lesions would destroy in large part the nucleus ambiguus (the vagoglossopharyngeal nucleus), the roots of the vagus, the hypoglossal nucleus in part, the descending root of the glossopharyngeus, the nucleus of the column of Burdach, the substantia gelatinosa, and the descending root of the fifth. The symptoms would therefore be mainly those of glossopharyngeal, vagal, and hypoglossal disease, and disorders in the sensory distribution of the fifth. The fillet might be involved in either ventral or dorsal lesions, according to the manner in which these extended towards the centre of the postoblongata. Ventrolateral lesions of the third fourth of the postoblongata (*C*, Fig. 453, and transection shown in Fig. 457) would implicate chiefly the pyramidal tract, the lateral columns, the fillet, the twefth nerve, and, if a surface lesion, probably a portion of the spinal accessory root bundles. The symptoms would include contralateral paralysis of the extremities, probably some contralateral sensory paralysis, hypoglossal paralysis, and some evidences of spinal accessory disease. A dorsal lesion would involve the columns

and nuclei of Goll and Burdach, the nucleus of the hypoglossus, and the descending roots of the fifth and of the glossopharyngeus, the symptoms being chiefly referable to nuclear disease of the twelfth nerves, and sensory disorders in the distribution of the fifth and the ninth.

Lesions of the Sensory and Motor Decussations.—The lower fourth of the postoblongata (*D*, Figs. 453 and 454, and transection shown in Fig. 458) contains within its limits the sensory decussation (decussation of the fillet), and a transection through it, as shown in Fig. 458, reveals in the ventral portion the pyramidal tract, remains of the ventral horns of the cord, the decussating fibres of the fillet, and the descending root of the fifth. In the dorsal portion of the cord

FIG. 458.

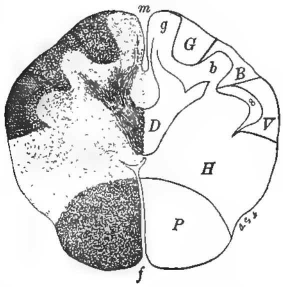


FIG. 459.

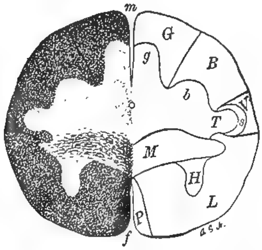


FIG. 458.—Transection of the postoblongata at the level of the sensory decussation (through *D* in Figs. 453 and 454): *g*, nucleus of the column of Goll (nucleus gracilis, nucleus clavatus); *G*, column of Goll (posteromedian column); *b*, nucleus of the column of Burdach (nucleus cuneatus); *B*, column of Burdach (posteroexternal column, posterolateral column); *s*, substantia gelatinosa; *V*, descending (spinal) root of the fifth (the tubercle of Rolando is formed chiefly by the descending root of the fifth, and the substantia gelatinosa); *D*, sensory decussation; *H*, substantia reticularis grisea (largely constituted of cinerea which at lower levels forms the ventral horn; the median portion of this is known as the substantia formatio reticularis alba, and consists almost entirely of medullated fibres); *P*, pyramid; *f*, anterior median fissure; *m*, fourth ventricle.

FIG. 459.—Transection at the level of the pyramidal (motor) decussation: *M*, motor decussation; *T*, remains of the posterior horn; *H*, remains of the ventral horn; *L*, anterolateral column. (The other letters indicate the same structures as in Fig. 458.)

are the nucleus and columns of Goll and Burdach, the nucleus of the spinal accessory nerve, and the descending roots of the fifth and ninth nerves. A unilateral lesion in this position would probably involve more or less both ventral and lateral or dorsal and lateral portions of the postoblongata, and the sensory symptoms might be of a somewhat confusing kind so far as the two halves of the body are concerned, although the motor manifestations, if the lesion was entirely above the motor decussation, would be paralysis of the extremities of the opposite side. Symptoms referable to the descending root of the fifth would still be prominent, as they have been in all levels of the postoblongata. A section (Fig. 459) along the line *e* (Fig. 454) would traverse the motor decussation and give some paralytic symptoms on both sides of the body, with also confusing sensory

symptoms. Symptoms showing involvement of the descending root of the fifth and of the nucleus and root bundles of the spinal accessory might be present. The descending glossopharyngeal root also extends below the motor decussation and might be involved in a lesion at this level. If the lesion extended somewhat caudad the roots of the first cervical nerves might be implicated.

Cases showing the Effects of Lesions of the Postoblongata and Oblongata-Spinal Transition.—*General Remarks.* While the recorded cases illustrating the effects of small gross lesions of the postoblongata and oblongata-spinal transition are not numerous, they are instructive and bear out the rules and principles of diagnosis which have been presented in the preceding paragraphs. When discussing the anatomy and physiology of the trigeminus, reference was made to the experiments of Turner on the tubercle of Rolando; and it will be remembered that its destruction caused loss of all forms of sensibility in the distribution of the trigeminus, and loss of the tactile sense in the body and limbs on the side of the lesion, with at the same time loss of the pain sense on the opposite side. Changes in the pupil, narrowing of the palpebral fissure, and diminished prominence of the eyeball, on the same side, and paresis of the arm and leg of the opposite side, were other results (page 859). A case recorded by Stieglitz, in which Turner believed that the symptoms suggested a lesion in close proximity to the tubercle of Rolando, was referred to when considering trigeminal anesthesia from oblongatal lesions (page 884). It is scarcely necessary to recall here that the tubercle of Rolando is a rounded mass situated laterally at the extreme lower end of the postoblongata (page 74). It is therefore a good anatomical landmark in the discussion of a subject like the one under consideration. Lesions of this tubercle and its vicinity should give symptoms showing implication of the clavate and cuneate nuclei, of the great spinal root of the fifth, and of both the sensory (fillet) decussation and the motor (pyramidal) decussation. In one case, recorded by Bechterew and cited by Turner, the symptoms were observed one year after a bullet wound was inflicted on the left side at the level of the atlas; on the right side there were anesthesia of the face, paralysis eventually becoming merely weakness of the right arm and leg, dulling of the sense of touch, and complete palsy of the "muscle-sense." On the left side were paresis of the arm and leg, which eventually disappeared, loss of the senses of pain and temperature, and dulling of the sense of touch, but retention of the "muscle-sense." The lesion was probably not completely unilateral, a fact which, as Turner suggests, may account for the dulling of tactile sensibility on both sides. A definition is not given of the term "muscle-sense," but "if it may be interpreted as the sense of position, then its retention on the side on which tactile sensibility was least affected, and association with loss of painful sensibility, is in close harmony with

the recorded experiments upon the Rolandic tubercle." (Turner.) Some years since I was called in consultation to see a man beyond middle age, a member of an apoplectic family, several of his near relatives having died from encephalic hemorrhage. He had had a sudden and severe apoplectic attack, from which he had partially rallied at the time of my visit. At first he suffered from difficulty in swallowing, dyspnea, and irregular action of the heart, but these symptoms had gradually become less severe. He had almost continuous hiccoughs. He was anesthetic on one side of the face, and partially so in the limbs of the same side. The pupil on the same side was dilated. He was unable to stand, having some loss of power on both sides, but much more marked on one. A curious symptom, but one which may have its explanation in the site of the lesion, was that the patient was constantly troubled with the feeling that his exact double was alongside of him in bed. This impression was of the most vivid character. The diagnosis at which I arrived was that he had had an attack of hemorrhage, probably from the vertebral artery or one of its branches; that this hemorrhage was not in the substance of the oblongata or the cord, but was probably subdural; and that the effusion had compressed and irritated various structures about the junction of the postoblongata and spinal cord,—the lower cranial nerves, the tubercle of Rolando (and therefore the descending root of the fifth), both the sensory and the motor decussation, and the cervical nerves, especially the phrenic. The hiccough was probably due to involvement of the phrenic nerve or its centre. The belief that he was physically duplicated may in some way have had its origin in the peculiar manner in which the lesion affected either the motor or the sensory decussation, or both, so as to call out an unusual dual action of the brain. The patient eventually made a partial recovery, regaining his health so far that he was able to go about.

Tumors of the Postoblongata and Oblongata-Spinal Transition. Glynn has reported a case of a very small tumor situated at the extreme lower portion of the postoblongata. The patient had loss of power in both legs and arms and difficulty in breathing, these being followed by numbness in the fingers of the right hand, and later by numbness in the calves, and then by weakness of the legs and arms, especially of the right arm. The tongue was swollen and numb. Cutaneous sensation was impaired over both arms, the sight was dim, the pupils were somewhat dilated but equally so, and the voice was thick and weak, apparently because of defective action of the tongue, although it was protruded naturally and in a straight line. The soft palate was not paralyzed, but the reflex was absent. The patient had some difficulty in swallowing when lying down, but did not regurgitate through the nose. The plantar reflexes were normal on the right side, but exaggerated on the left and delayed, with a tendency to

cross, the left foot moving when the right was tickled. The patellar reflex was absent, the cremasteric was normal. The patient could not straighten his legs when they were flexed at the knee, and there was considerable loss of power in his hands and in the muscles of the trunk. At the autopsy a gliomatous tumor was found in the middle line of the postoblongata at the line of the calamus scriptorius. It was not larger than a pea, and it was placed about one eighteenth of an inch from the posterior surface of the postoblongata. Osler records a case of a patient with a history of syphilis who had suffered for a long time with headache and then began to have fits. The headache was chiefly occipital. The patient was unsteady in walking, and had numbness, tingling and crawling sensations in the hands and feet, and stiffness and pain in the muscles of the back of the neck. The autopsy showed a glioma in the lower part of the postoblongata and the beginning of the cord. In this case the symptoms pointed to implication of both the sensory and the motor decussation and to accessorius involvement. Gliomatous tumors frequently involve structures without destroying them to such an extent as to interfere completely with their functions, which probably accounts for this case not having produced even more marked symptoms. In a case recorded by Weir autopsy showed a grayish translucent tumor, which proved to be a spindle-celled sarcoma, on the under surface of the left lobe of the cerebellum, which from its position had exerted considerable pressure on the oblongata, crowding it to the right and forward. The growth had also invaded the fourth ventricle and had extended downward between the dura and the pia for a distance of ten centimetres to the left lateral posterior region of the spinal canal, the cord being crowded forward and to the right. The growth seemed to have originated in the pia and had no connection with the substance of the cerebellum. The chief focal symptoms were precipitate micturition and defecation ; cramps of the left calf ; later, clonic spasms of the left leg ; cramp and numbness of the left hand, and still later the same in the right hand ; sinking of the left eyeball and contraction of the pupil ; paralysis of the left biceps, triceps, and deltoid ; paresis of the left leg which slowly developed to more complete paralysis ; and exaggerated knee jerk on the left side. The patient also had intense frontal headache daily on the right side, and slight left optic neuritis. Shortly before death he was attacked with twitchings on the right side of the body, and suddenly became stupid. This patient was trephined over the motor region of the cortex, most of the symptoms being motor and in the limbs of the left side. The particular symptoms which should have assisted in a more exact diagnosis of the site of the lesion were the precipitate micturition with defecation, the contraction of the pupil and sinking of the eyeball, and the paralysis of the biceps, triceps, and deltoid. These phenomena clearly pointed

to the lower portion of the oblongata and the upper portion of the cord, although the others were just as readily explained by a lesion of the motor region of the cerebrum.

Abscess of the Postoblongata. Abscess of the postoblongata is a rare affection, but an interesting case has been recorded by Eisenlohr. The patient complained of stiffness of the left arm, and the arm and leg of this side became paretic, with reduced sensation; later the arm and hand were almost completely anesthetic, with increase of the paralysis, and still later the right side became involved. Signs of implication of the facial, hypoglossal, or ocular nerves were not at any time present. Autopsy showed, in addition to an empyema cavity, a gangrenous cavity of the size of an apple in the left lung, at a level with the fifth rib anteriorly. On the left side of the floor of the fourth ventricle, in the region of the ala cinerea, an abscess as large as a pea was found.

Pseudohydrophobia due to Lesions of the Oblongata-Spinal Transition. Whatever views may be taken as to the occurrence of hydrophobia in the human being, no doubt can exist that many of the cases called "hydrophobic" and attributed to the action of a specific virus can be better explained in other ways. I have seen in consultation or otherwise cases in which the symptom picture was typically or almost typically that which is given in the books as that of hydrophobia, but in which either postmortem findings or the subsequent progress of the case demonstrated that the clinical phenomena could be much better explained on some other hypothesis, such as that of a small focal lesion. In a case recorded by me, the patient was a child only two and a half years old, who several months before the attack had been bitten by a Spitz dog. The little patient suffered, among other symptoms, from pharyngeal spasm on slight peripheral or mental excitation. The autopsy in this case revealed a pachymeningitis of considerable extent just at the junction of the oblongata and the cord. With Dr. Charles Hoban, of Philadelphia, I saw another case supposed at the time to be one of hydrophobia. The patient was a child five years old who, seven months before he was taken sick, had been bitten in the hand by a dog. He first complained of soreness in the hand and wrist on the side that had been bitten, and the hand of this side became paralyzed. When first seen by me the arm was completely paralyzed, and the patient complained of some pain in the shoulder and neck, was extremely restless, and refused to take medicine or food. Efforts to take either food or drink brought on laryngeal and pharyngeal spasm. The paralysis of the arm continued, but he had no pain. He had several convulsive attacks, and his lungs became edematous; no sensory disorders were present. He died apparently of cardiac and respiratory paralysis. This would seem to be a case in which there was a small irritative and destructive lesion just above the pyramidal decussation.

BULBAR PARALYSIS.

Synonyms and Varieties.—Having considered separately the diseases of the cranial nerves, it will be necessary, in order to complete the survey of this portion of the nervous system, to direct attention to *bulbar paralysis*, a disease which may involve all or nearly all the nerves from the twelfth to the third. Several forms of true bulbar paralysis are generally recognized, as *acute bulbar paralysis*, *chronic progressive bulbar paralysis*, and *asthenic bulbar paralysis*. Pseudobulbar paralysis, discussed in Chapter IV. (page 353), is not a true bulbar affection. By far the most common form of this disease is the chronic progressive bulbar paralysis which has been known by names as numerous as have been the ideas regarding its pathology and the special methods of grouping its symptoms. It was called by Duchenne, to whom our first exact knowledge of the disease is due, "paralysis of the tongue, the soft palate, and the lips;" other synonyms in common use are atrophic bulbar paralysis, bulbar nuclear paralysis, and glossolabiolaryngopharyngeal paralysis, or primary labioglossolaryngeal paralysis. In one of its forms it is the polioencephalitis inferior chronica of Wernicke.

ACUTE BULBAR PARALYSIS.

Acute bulbar paralysis can be dismissed here with a few words. In some of its types it has already been described (see acute polioencephalitis superior and inferior of Wernicke, page 531; also the discussion of acute ophthalmoplegia, page 834). Under the more general name of *acute bulbar myelitis*, types of this disease which cannot be strictly classed with either the inferior or the superior form of polioencephalitis are sometimes seen; but the mistake must not be made of supposing that there is a distinct form of acute bulbar paralysis which bears no pathological relationship to these types of polioencephalitis. This affection usually begins with headache, vomiting, and vertigo, difficulties in articulation and deglutition soon appearing. Persistent hiccough is another frequent symptom. The muscles supplied by the cranial nerves are successively but rapidly paralyzed. Commonly the muscles of the lower part of the face, of the tongue, palate, and pharynx, are among the first to be attacked. The ocular muscles may or may not be involved in the attack, and ophthalmoplegia when present is usually partial, and is most likely to show itself by implication of both abducent nerves. Disorders of respiration and cardiac action appear early. The extremities may or may not be paralyzed. The rectum and bladder are commonly paralyzed towards the end of the disease. The patient, retaining consciousness until nearly the end, usually dies cyanosed and asphyxiated. The disease has its real pathological cause in changes in the vessels which supply the nuclear regions attacked. Its diagnosis is readily made if the

succession of symptoms described is borne in mind. The disease is almost invariably rapidly fatal. The only treatment consists in complete rest and removal of all sources of excitement, with perhaps remedies like strychnine and atropine to support the heart and the respiration.

CHRONIC PROGRESSIVE BULBAR PARALYSIS.

Symptomatology.—The disease usually begins so insidiously that it is well advanced, so far as the nuclear degeneration is concerned, before it is clearly recognized by the patient or his friends. Premonitory symptoms, such as pain in the head and neck and vertigo, may be present, but more frequently no history of such symptoms is given. Almost invariably the first symptoms to attract attention are difficulties in enunciation and intonation: the patient finds that he has a little trouble in pronouncing words containing the lingual sounds, such as *l*, *r*, and long *e*. Soon interference with all forms of articulation is evident. The patient now has difficulty with other linguals, and with the labials, such as *a*, *o*, *u*, *p*, *b*, and *v*. Swallowing is often impaired, even at an early period, and a little later regurgitation, choking, and coughing may be distressing and dangerous symptoms, these being due to the progressive implication of the pharyngeal and palatal muscles. The tone of the voice becomes nasal, and vocalization difficult and labored. As the disease progresses, the impairment of the movements of the tongue, palate, and pharynx becomes more marked, and the muscles of the facial supply are more and more involved. This results in a change in the appearance of the face, so that the patient shows a want of expression in the lower part of his face which is emphasized by the retention of ocular movements and of movements of such muscles as the frontales and corrugators. It assumes a half masked appearance, which is at times disturbed by waves of tremulous movements the result of efforts at innervation of the weakened muscles. Movements requiring skilful and decisive use of special muscular groups are markedly interfered with; the patient, for instance, is soon unable to whistle, or to pucker the mouth as in kissing; he cannot protrude the tongue, or can protrude it only very imperfectly; while late in the disease it may lie a shrunken, corrugated, inert mass on the floor of the mouth. While the disease has a method of progression which is more typical for the vast majority of cases than that for other degenerative diseases, occasionally its usual course is modified to a greater or less extent. Instead of the muscles supplied by the hypoglossus being the first attacked, it may happen that the laryngeal muscles or even the facial muscles first show evidences of the disease; but such a method of onset is rare. As the disease progresses, those portions of the vagus nucleus which preside over the innervation of the lungs and heart may become so much involved as

to cause the death of the patient from a true respiratory or cardiac paralysis. More frequently, however, death is due to general inanition or exhaustion, and such an end comes sooner if the patient is not fed artificially. The masseter and other muscles in the motor distribution of the trigeminus are attacked in comparatively rare instances, usually the pterygoids being the first of the trigeminal group to be attacked. Difficulties in deglutition and mastication are much more commonly, for a lengthy period at least, due to the impairment of the tongue and of the accessory muscles of mastication, namely, those of the palate, pharynx, and facial supply. The ocular muscles may or may not become involved in the disease. Probably the degenerative process attacks to a limited extent the nuclei of the muscles of ocular movement in the majority of cases. Drooling is one of the most frequent, striking, and annoying symptoms. This is in part due to the paresis of the oral and facial muscles, allowing the saliva to escape from the corner of the mouth, but in part it is probably dependent upon an increase in the actual amount of saliva secreted. Fibrillary contractions are usually present at some period of the disease, but most frequently in the later stages, except perhaps in the tongue, where close investigation may reveal their presence even at a comparatively early period. Late they are marked in the tongue, and may also be observed in the lips and sometimes in the muscles of the face. In the terminal stage of the disease the patient has increasing difficulties in swallowing; food collects in the mouth and threatens his life by choking. If the esophagus becomes involved in the paralysis, deglutition is absolutely impossible, and he must be allowed to starve or be fed with the stomach tube. Even long before this stage is reached he secures food with any approach to comfort only by this method of artificial feeding. Atrophy sooner or later is marked, and is especially noticeable in the tongue and lips. Palatal, pharyngeal, and laryngeal reflexes disappear. At a very late period not only may respiration and cardiac action be interfered with directly, but the accessory muscles of respiration, those supplied by the spinal accessory, phrenic, and thoracic nerves, may be impaired or may cease almost entirely to act. The reactions of degeneration, and especially partial degeneration reactions, can be obtained in accessible muscles. In typical cases sensibility is not affected, although some disturbance of sensation in the trigeminal supply has been noted. Tinnitus and gradual impairment of hearing are other infrequent phenomena. The mentality of the patient remains good, except so far as it may be impaired by the constant strain upon his emotional nature and the lack of proper cerebral nutrition due to the inability to take sufficient food. Sooner or later great emaciation occurs.

Pathology.—The pathological anatomy of bulbar paralysis is perhaps as well understood as any other form of chronic degenera-

tion, unless it be tabes and paralytic dementia. Primary atrophy of the large multipolar ganglionic cells of the motor nuclei of the bulb is the underlying pathological condition. Specimens examined from patients who have died from intercurrent affections, at different stages of the disease, show that the amount of this primary atrophy is coextensive with the symptomatology of the disease. While innumerable pathological examinations have been made since the days of Duchenne, there has been little advance in our knowledge of its pathology since he announced that it was due to a primary pigmentary degeneration and atrophy of the larger ganglionic cells of the nuclei of the hypoglossal, pneumogastric, glossopharyngeal, and other nerves involved.

Diagnosis.—The diagnosis of chronic progressive bulbar paralysis is not difficult, although it is closely simulated by several other well known paralytic disorders. The most important help is given by a critical study of the method of initiation of the disease and the peculiar succession of its symptoms. The two affections from which it needs to be most frequently distinguished are cerebral pseudobulbar paralysis and asthenic bulbar paralysis. Cerebral pseudobulbar paralysis has been considered in Chapter IV., page 353. In pseudobulbar paralysis apoplectic attacks may recur, and the disease instead of advancing steadily and progressively is more likely to be augmented by paroxysmal increments. One of the most important points of distinction between the true bulbar paralysis and the pseudobulbar paralysis is the condition of the tongue, which in the former becomes markedly atrophied and is the seat of fibrillary contractions, from which in the latter it is free. The diagnosis of asthenic bulbar paralysis will be considered in the next section. Gross lesions of the oblongata may in some instances somewhat closely counterfeit progressive bulbar paralysis. A glioma, sarcoma, or gumma, gradually increasing in size and infiltrating the substance of the bulb, may cause a chain of symptoms similar in their general features and in some special characteristics. A growth or any form of gross lesion, however, is not likely to implicate both sides of the oblongata with the same uniformity and in the same method of progression as chronic processes of nuclear degeneration.

Prognosis.—From the very nature of the disease the prognosis as to life is unfavorable. No case of true chronic progressive bulbar paralysis has ever recovered, although recovery has taken place in some of the diseases which simulate and counterfeit it. The prognosis as to duration and time of fatal issue is variable. Now and then a case is comparatively rapid in its downward course, the patient dying in from one to two years; more commonly he lives for four or five or more years.

Treatment.—From what has been said about the course and prognosis of bulbar paralysis it follows that treatment is of no avail

in stopping the progress of the disease to a fatal issue. Probably something can be done to retard the advance of the affection, mostly by measures calculated to improve the general nutrition of the patient. Physical and mental rest, rest of the organs implicated in the advancing disease, remedies which promote nutrition, like cod liver oil and concentrated foods, travel and recreation which do not involve too much muscular activity, are all measures of value in maintaining the patient's general condition and in staying the rapid progress of the disease. The use of moderate faradic and galvanic currents to the face, tongue, pharynx, and larynx seems to do temporary good, but electricity should not be used too frequently or too actively, as overuse and artificial excitation of muscles not properly innervated may hasten degeneration. Artificial feeding is a matter of the utmost importance in the late stages of bulbar paralysis.

ASTHENIC BULBAR PARALYSIS.

Definition and Synonyms.—Under the name of asthenic bulbar paralysis, a disorder in some particulars closely simulating progressive bulbar paralysis has been described.* It is a disorder in which the symptom complex is almost exclusively motor and consists chiefly of symptoms which point to exhaustion of the centres for the motor cranial nerves, although all parts of the motor nervous system may take part in the disturbance. The speech and the swallowing mechanism are, as a rule, especially affected. The affection has been designated by various names in addition to that given at the head of this section, as bulbar paralysis without anatomical foundation, and *myasthenia gravis pseudo-paralytica.*

Symptomatology.—The symptoms of the disease are usually presented in paroxysms recurring at irregular intervals. The patient at first most commonly suffers from a feeling of fatigue on slight exertion, which may soon go on to the point of entire exhaustion, especially of his bulbar mechanism. Articulation and deglutition may be extremely impaired and the limbs show great weakness amounting almost to a true paresis. In the case recorded by Collins, as in others, the first noted symptoms were referable to the ocular muscles. The patient had ptosis first on one side and then on the other, and in both cases this was associated with double vision. Examination of the ocular muscles often shows no objective recognizable insufficiency or paralysis. When objective symptoms are present they are commonly bilateral and transient. Other symptoms present in the case of Collins were weakness of the masticatory muscles, defects in articulation and vocalization, unwieldiness in the movements of the

* An excellent recent paper on asthenic bulbar paralysis, freely used by the author, is by Dr. Joseph Collins, published in the International Medical Magazine, April, 1896.

tongue, and distressing attacks of tachycardia and dyspnea accompanied by a decided feeling of weakness and exhaustion in the muscles both of the trunk and of the extremities. Sometimes tremor of half of the face is present. The knee jerks are readily exhausted, but myotatic irritability remains normal. Sensation and electrical responses are unchanged, and various other negative conditions, pointing to the fact that the disease is not a true bulbar paralysis, will be given under diagnosis.

Etiology and Pathogenesis.—We have no positive knowledge regarding the etiology and pathogenesis of this affection. In most of the recorded cases the disease has occurred under the age of thirty years. Traumatisms, syphilis, emotional strain, physical and mental exhaustion, and various other of the causes so frequently assigned as the source of diseases of the central nervous system have apparently been present and active in some cases. Anemia and chlorosis have also been noted. The weight of opinion, although unsupported, is in favor of the toxic origin of the disease. It is generally held to be due to the presence in the system "of a poison, of exogenous or endogenous origin, which manifests its peccant action on groups of ganglionic cells exclusively motor in any part of the nervous system, but by preference those of the medulla and of the pons." (Collins.) Although a number of autopsies have been made on well observed cases, no pathological changes have as yet been found.

Diagnosis.—In the diagnosis of asthenic bulbar paralysis from progressive bulbar paralysis of organic origin it is first important to note the absence of certain clinical phenomena which are distinctive of the latter disease. In asthenic bulbar paralysis, drooling, fibrillary contractions, true atrophy of the affected muscles, disorders of common sensibility and of the special senses, alterations in electrical reactions, and changes in the deep reflexes are all absent. The patient in this affection recovers almost completely from the attacks, even when these are of the most pronounced character. The muscles supplied by the facial, motor fifth, and third nerves are more likely to be distinctly and largely although transiently affected in the asthenic than in the organic form of the disease. In the genuine progressive bulbar paralysis the hypoglossus, vagus, and glossopharyngeus are more markedly affected. It must not be forgotten, however, that in both diseases the association and sequence of phenomena, so far as peripheral cranial nerves are concerned, may vary greatly. Asthenic bulbar paralysis in several particulars simulates the cerebral pseudobulbar paralysis more closely than the latter counterfeits the organic progressive bulbar affection. In both, for instance, the patient is liable to have recurring attacks of paroxysms. In the cerebral pseudobulbar cases, however, these attacks are distinctly apoplectic and have residua of a decided character. Symptoms pointing to cerebral involvement, such as headache, dizziness, and

temporary mental confusion, are more common in the cerebral than in the asthenic bulbar type. In both of these affections the conditions as to atrophy, fibrillary contractions, and electrical response are the same. Multiple paralysis of the cranial nerves from associated neuritis is a rare affection, the diagnosis of which from asthenic bulbar paralysis of cerebral type is to be made chiefly by the presence, in the disease due to neuritis, of degenerative electrical reactions, true atrophy, and in some instances by the presence of phenomena of sensory irritation. Owing to the frequent occurrence of tachycardia, asthenic bulbar paralysis may perhaps be regarded as an aberrant or abortive form of exophthalmic goitre; but the diagnosis of this affection will be cleared up by a study of the course and special clinical phenomena of the former. I have seen a few cases of gliosis in its early stages attacking the bulb, and other cases because of their history presumably of syphilitic infiltration or gummatous meningitis, which have closely simulated the phenomena and progress of asthenic bulbar paralysis as usually described. The cases of gliomatosis eventually develop other decided symptoms of syringomyelia, and those of gummatous meningitis or syphilitic infiltration of the substance of the bulb improve rapidly under specific treatment, unless allowed to go long without any therapeutic interference. The syphilitic cases in particular do not, as a rule, have the other evidences of general myasthenia. In rare cases hysteria or hysteroneurasthenia may present phenomena which somewhat closely simulate those of asthenic bulbar paralysis.

Prognosis.—The prognosis of asthenic bulbar paralysis varies according as the type of the disease is severe or mild. In the severe cases it leads rapidly to death,—more rapidly, in fact, than in cases of the organic type. In other cases the patients may have a number of attacks making complete or nearly complete recovery from all. The average duration of the disease is less than that of progressive bulbar paralysis. It usually lasts from two to four years. Cases of complete recovery have been recorded.

Treatment.—The most important therapeutic measures are rest, careful feeding, and the removal of sources of irritation and anxiety. Collins believes that artificial feeding by means of the stomach tube should not be resorted to, as the movements of regurgitation produced by the passage of the tube are more exhausting to the patient than is the act of swallowing artificially masticated and liquid food. Central galvanization is of temporary service, and the general nutrition of the patient may be improved by hydrotherapy. The local use of electricity to the throat, larynx, tongue, and other parts affected is calculated to do harm rather than good, by exciting the already weakened nerve centres. The hypodermatic use of strychnine proved of value in the hands of Collins. The metallic tonics and nutrients like malt and cod liver oil are of value.

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